The New England
Journal of Medicine

Copyright, 1943, by the Massachusetts Medical Society

VOLUME 228 JUNE 10, 1943 NUMBER 23

COMPOUND FRACTURES OF THE SKULL*

The Results of Surgical Therapy in Two Hundred and Eighteen Cases

DONALD MUNRO, M.D.†

BOSTON

WITH the advent of the sulfonamide group of drugs there has been a tendency among members of the medical profession to discard the results of the studies that have been made in previous years along other lines, and to substitute chemotherapy in their place. Since the entrance of the United States into the war, when the need for dealing with battle and air-raid casualties arose, this tendency has been particularly apparent in the field of surgery. This, together with the ill-considered lay-magazine and newspaper publicity that followed the first reports of the neurosurgical and other casualties at Pearl Harbor, calls for a critical analysis of what properly conceived surgery is able to accomplish without the aid of chemotherapy. The object of this analysis is to discover whether chemotherapy can justifiably be expected to be of assistance in this problem, and whether the indiscriminate use of chemotherapy may do harm even beyond the point of leading to the neglect of fundamental surgical principles that have successfully stood the test of time. The need for this knowledge is especially acute in traumatic craniocerebral surgery, and in particular in compound fractures of the skull. Of necessity, the great majority of these cases will have to be cared for by general surgeons who are not experienced in this field. They appreciate their lack of experience and the deficiencies that go with that lack more keenly than anyone else, and are only too eager to profit from the experiences and mistakes of others.

The data presented herewith, together with a review of certain pertinent articles in the English and Canadian literature, are the result of eleven years' experience with patients suffering from injuries to the central nervous system. They are presented chiefly to emphasize that although chemotherapy with the sulfonamides is indispensable, it cannot and should never be used as a substitute for properly conceived surgery in the treatment of compound fractures of the skull.

Up to January 1, 1942, 218 cases of all types of compound fracture of the skull have passed through my hands. These were divided as follows: 29 involved one or more paranasal air sinuses but had no cerebrospinal rhinorrhea; 12 patients had cerebrospinal rhinorrhea, either with or without involvement of a paranasal air sinus; 16 had bullet wounds; 6 had perforated wounds; 2 had a complicating avulsion of the scalp; and 153 had simple compound fractures.

In addition to the compound fractures of the skull, 23 patients had major injuries in other parts of the body. Twenty of these had fractures of other bones as follows: lower extremity, 9 cases (pelvis, 2; femur, 2—one compounded and accompanied by a fractured ilium; both bones of the lower leg, 3—one comminuted into the knee joint; tibia, 1; and fibula, 1); upper extremity, 9 (clavicle, 2; humerus, 1; both bones of the forearm, 2—one accompanied by a fracture of the olecranon of the other arm; radius, 1; and Colles's fracture, 3—one comminuted into the wrist joint); spine, 1; and facial bones, 1. Three patients had multiple injuries, all accompanied by a ruptured lung and 1 by edema of the glottis in addition.

Sixteen patients had other severe associated cranial or intracranial damage. Three had multiple compound fractures of the skull, one accompanied by exsanguination, and 2 had crushed skulls, one in addition to other injuries. Three patients had ruptured cranial venous sinuses, one with an extradural hematoma. Five had subdural hematomas, one with an extradural clot. There were 3 cases of severe ocular injury. In two of these one optic nerve was completely severed, and in one the other nerve was also par-

*From the Department of Neurosurgery, Boston City Hospital.

†Assistant professor of neurosurgery, Harvard Medical School; associate professor of neurosurgery, Boston University School of Medicine, and surgeon-in-chief for neurosurgery, Boston City Hospital.

tially severed; the third patient had a hemorrhage into the anterior chamber of one eye. Other complicating conditions were: syphilis, 4 cases, acute alcoholism, 3; severe heart disease, 2; toxic dehydration, 2; and miscellaneous conditions, 11—1 case each of intracranial tumor, decerebration, epilepsy, carotid-artery and jugular-bulb fistula, osteomyelitis of skull, psychosis, puncture wound of the right knee, gas-bacillus infection of a wound of refused operation. In 3 others it is not clear from the record what the reason was. Only in the remaining 7 (12 per cent) did the surgeon have a legitimate opportunity to exercise his judgment and elect not to operate. In two of these 7, the compounded fracture involved the anterior wall of one frontal sinus only, in another two there was a complicating extensive avulsion of the scalp, and in one the fracture was compounded directly over

TABLE I. *Age Incidence.*

AGE	TYPE OF COMPOUND FRACTURE						
	SIMPLE	INVOLVING PARANASAL SINUSES, WITHOUT RHINORRHEA	INVOLVING PARANASAL SINUSES, WITH RHINORRHEA	WITH AVULSION OF SCALP	BULLET WOUNDS	PERFORATING WOUNDS	TOTAL
yr.							
Under 10	47	1	4		1	2	55
10–19	20	1	1		3	1	26
20–29	21	6	2	1	4	1	35
30–39	17	9			1		27
40–49	12	8	2	1	3		26
50–59	19	1			1	2	23
60–69	7	2	1				10
70 or over	4	1	1		1		7
Not known	6		1		2		9
Totals	153	29	12	2	16	6	218

the leg, pyelitis, pregnancy and inoperable carcinoma of the rectum.

All age groups were represented, the youngest patient being three months old and the oldest eighty-one years (Table 1). The largest single group (55 cases) was made up of children under ten years of age. Among them were 26 that were five years old or younger. Seven patients were over seventy.

Sixty of these 218 patients died, a mortality of 27.6 per cent (Table 2). There were one hundred and fifty-nine operations—an operability rate of 73 per cent. No patient was operated on more than once. Twenty-five of these 159 patients died—an operative mortality of 15.7 per cent. In contrast to this, there were 59 patients who were not operated on. Thirty-five of this group died—a mortality of 59.3 per cent. A study of the reasons that lead to a decision not to operate indicates that not only these injuries but those in all the patients were at least as severe as, and probably severer than, any average similar group. Operation was decided against because of surgical shock in 27 cases, poor general condition in 7, other complicating major injuries or infection in 5, and because the patient was admitted too late in 5. Thus, 44 (75 per cent) of the 59 patients were not operated on because of adequate reasons that were beyond the control of the surgeon. In addition, 4 patients were not operated on because the diagnosis was missed, and 1 because the patient the superior sagittal sinus. In a patient with cerebrospinal rhinorrhea, the fistula closed spontaneously in twenty-four hours, and in another, chemotherapy was substituted for surgery.

It is obvious that in this series, at least, the presence of surgical shock and analogous allied conditions constituted a major factor in reaching a decision concerning operability. By the same token, these same conditions played a most important part in determining the length of time that had of necessity to be allowed to elapse after the infliction of the injury and before operation could be undertaken without prohibitive risk.

Four principles have governed the therapy of compound fractures of the skull in my clinic. The first is that all compound fractures of the skull must be completely débrided within forty-eight hours of the infliction of the injury, or else not operated on until the wound has been completely healed for six to eight months, depending on the type and degree of infection. The second principle is that no patient shall be operated on until he is out of surgical shock and until his general condition warrants it. The third is that after the diagnosis has been made by palpation through the wound by the surgeon's sterilized finger, the first dressing shall be one that can be applied with an absolute minimum of handling of the wound. The fourth is that the débridement shall be complete unless there is a communication with the nose or involvement of the supraorbital ridge, shall be done

in such a way as to avoid spreading the bacterial contamination throughout the wound or producing tissue necrosis through tight sutures, and shall include the removal of all large foreign bodies. No wound that has been properly and therefore completely débrided has been drained. In all undrained wounds, unless the wound heals by first intention and without evidence of infection of the scalp, bone, meninges or brain, it is considered to be a septic one and the result to be unsatisfactory. Drained wounds are not expected to heal by first intention or necessarily to be free of meningeal infection. Deaths caused by postoperative sepsis in any form are not considered justifiable, and an attempt is made in every such case, as well as in every case of postoperative sepsis following a drained or undrained wound, to determine the proximate cause of the sepsis and death.

The first and second principles noted above have to do with determining the optimum time of operation. There can be no doubt that such a major procedure as débridement of a compound fracture of the skull should not be attempted until the patient is out of surgical shock and until his general condition is such as to do away with any risk that prohibits operation. It is apparent that there is a wide variation in the severity of the compound fractures of the skull seen in different neurosurgical clinics, and that those surgeons who see a small percentage of severe injuries are prone to operate early. It is axiomatic, of course, that the earlier one can débride such a wound, the better the chances of preventing the contamination in the wound from becoming an infection. This axiom holds good, however, only if the increased risk of death without infection as the result of a relatively immediate operation is properly assessed against the increased risk of infection without death from a postponed operation. The crux of this problem lies in the speed with which a contaminated wound metamorphoses into an infected wound in the absence of any extraneous factor, such as manipulation or operation. In the absence of evidence to the contrary, and on the basis of experience collected previous to the present war in analogous lesions of the long bones, this time has commonly been set at six to eight hours. My experience has convinced me that this period is needlessly short from the point of view of the bacterial content of the wound of compound fractures of the skull, and dangerously short from the point of view of risk to the patient's life.

Work by Miles,[1] Spooner,[2] Hare and Willits,[3] Smith[4] and others, moreover, suggests strongly that from the bacteriologic aspect the incidence of infection, as opposed to contamination of compounded wounds of the long bones and extremities, is unexpectedly low during the first forty-eight hours after infliction, and that much more im-

TABLE 2. *Mortality and Sepsis Rates.*

Type of Compound Fracture	All Cases				Unoperated Cases			
	Total	Deaths	24-hr. deaths	Mortality	Total	Deaths	24-hr. deaths	Mortality
				%				%
Simple	153	40	23	26.1	41	28	23	66.6
Involving paranasal sinuses, without rhinorrhea	29	11	0	38.0	2	0	0	0
Involving paranasal sinuses, with rhinorrhea	12	2	0	16.6	3	1	0	33.3
Bullet wounds	16	7	6	43.7	9	6	6	66.6
Perforating wounds	6	0	0	0	2	0	0	0
Avulsion of the scalp	2	0	0	0	2	0	0	0
Totals	218	60	29	27.6	59	35	29	59.3

Type of Compound Fracture	All Operated Cases					Adjusted Operated Cases*				
	Total	Deaths	Mortality	Septic cases	Percentage of sepsis	Total	Deaths	Mortality	Septic cases	Percentage of sepsis
			%		%			%		%
Simple	112	12	10.7	31	27.6	91	4	4.4	6	6.6
Involving paranasal sinuses, without rhinorrhea	27	11	27.6	6	27.2	21	6	28.5	2	9.5
Involving paranasal sinuses, with rhinorrhea	9	1	11.1	0	0	9	1	11.1	1	11.1
Bullet wounds	7	1	14.2	1	14.2	6	1	16.6	1	16.6
Perforating wounds	4	0	0	1	25.0	3	0	0	0	0
Avulsion of the scalp	0	0	0	0	0	0	0	0	0	0
Totals	159	25	15.7	39	24.5	130	12	9.2	10	7.6

*Figures corrected for technical errors.

portant factors in the control of wound infection in such cases are the immediate and as nearly as possible complete immobilization of the wound and the avoidance of cross infections from attendants, including physicians and nurses, other patients, dressings and bed linen. The importance of these sources of infection has also been emphasized by Trueta[5] and Barnes.[6] Cairns[7] has stated in relation to craniocerebral injuries that patients should be operated on as soon as the patient's general condition permits major surgery to be performed with safety. Otherwise the débridement may be postponed up to forty-eight hours after infliction, but no longer without materially increasing the risk of changing contamination into infection of the wound and thus producing postoperative sepsis. It is during this postponement that chemotherapy by one of the sulfonamide compounds proves to be of great value. If administered promptly and in adequate dosage by mouth and sprinkled in and about the wound at the earliest possible moment without manipulation of the latter, its bacteriostatic power will further guarantee the patient against wound infection during any necessary waiting period previous to operation.

Table 3. *Organisms Recovered from Wounds of Compound Skull Fractures.*

Organism	Simple Compound Fractures			Compound Fractures Involving Paranasal Sinuses without Rhinorrhea			Compound Fractures Involving Paranasal Sinuses with Rhinorrhea			Bullet Wounds			Perforating Wounds			All Fractures		
	Recovered Cases	Fatal Cases	Total Cases	Recovered Cases	Fatal Cases	Total Cases	Recovered Cases	Fatal Cases	Total Cases	Recovered Cases	Fatal Cases	Total Cases	Recovered Cases	Fatal Cases	Total Cases	Recovered Cases	Fatal Cases	Total Cases
Str. haemolyticus	1	2	3	0	0	0	0	0	0	0	0	0	0	0	0	1	2	3
Str. haemolyticus and *Staph. aureus*	2	1	3	0	1	1	1	0	1	0	0	0	0	0	0	3	2	5
Staph. aureus	21	0	21	0	1	1	2	0	2	2	2	4	1	0	1	26	3	29
Staph. albus	7	0	7	0	0	0	0	0	0	0	0	0	0	0	0	7	0	7
Meningococcus	0	0	0	0	1	1	0	0	0	0	0	0	0	0	0	0	1	1
No growth	9	0	9	1	0	1	1	0	1	0	0	0	0	0	0	11	0	11
Totals	40	3	43	1	3	4	4	0	4	2	2	4	1	0	1	48	8	56

do better if operation is postponed until their general condition is satisfactory and they have been transported to a hospital where adequate facilities are at hand. This has also been the Continental experience, especially in regard to postponement pending the availability of proper equipment and personnel, as evidenced by Jentzer[8] and Tönnis.[9]

I[10] recently made a study of the bacteriology of compound fractures of the skull in 52 patients (Table 3). Although all such wounds were considered to be contaminated at the time of infliction, positive cultures of pathogenic bacteria were obtained from only 55 per cent of cases in the first twenty-four hours and from only 66 per cent in the first forty-eight hours after infliction. On the other hand, pathogenic bacteria were cultured from 91 per cent of the wounds swabbed later than forty-eight hours after infliction. I have been unable to find other similar bacteriologic studies of compound fractures of the skull. I am therefore convinced that, pending more complete evidence, although all compound fractures of the skull are probably contaminated at the time of infliction, the degree of contamination is so small that in the absence of manipulation of the wound, cultures can be grown from only half the wounds in the first twenty-four hours and from only two thirds of them in the first forty-eight hours. It seems only reasonable to conclude that in the presence of such a small increase in culturability, excision of the contaminating bacteria can be postponed if necessary up to forty-eight hours after infliction of the wound without significantly increasing the risk of wound infection. On the other hand, because of the high rate of culturability (91 per cent), operations performed on these wounds after the first forty-eight hours following infliction have a prohibitive and unjustifiably high rate of infection. As stated above, if the wound is not manipulated, compound fractures of the skull

The third and fourth principles noted above are concerned with the avoidance of measures that spread the contaminating bacteria to hitherto uninvaded parts of the wound. They apply to the preoperative period as well as to the operation itself. Attention has been called to the fact that modern experience has demonstrated that immediate complete immobilization is the most important and essential guarantee against the spreading of infection in compound fractures and other wounds of the extremities. It has been shown that, aside from spreading by contact and the like, the lymphatics play the chief rôle in this dissemination.[6] In bruising wounds it is certainly theoreti-

cally possible, and indeed probable, that owing to the local crushing effect the lymphatics and small veins at the circumference of the bruised area are at once closed mechanically and that they remain so for a variable number of hours. During this period, the contaminating bacteria are locally static unless these veins and lymphatics are opened up by manipulation, or until sufficient bacterial growth has taken place to cause implantation of daughter cultures outside the limits of the contusion.

Mechanical transfer of bacteria to other hitherto uncontaminated areas can serve as another and important means of spreading infection. Such procedures as instrumentation and retraction of the tissues and irrigation of the wound act in this way. So, too, does palpation in the wound by the surgeon's finger. The evils of the latter procedure, however, are overbalanced by the advantages of a degree of diagnostic accuracy that cannot be attained in any other way. Although irrigation of the wound of a compound fracture of the skull is advised by many and countenanced by tradition, I am convinced that this is a dangerous procedure, whether done before or at operation. Moreover, since the same end can be accomplished in other and less dangerous ways, it has no justification in itself. The object of any irrigation is to direct a stream of fluid against the sides and bottom of a cavity with the intention of loosening unwanted débris from these areas, so that the stream returning from the depths of the wound will carry this material outside and away from the site of the injury. The greater the force and the larger the amount of irrigating fluid, the greater the cleansing effect. Where the walls and base of such a wound are solid and there is no communication with other cavities, all the ingoing fluid will return to the outside and the cleansing will be maximum and unaccompanied by any untoward side effects. This is the situation that exists in compound fractures of the long bones.

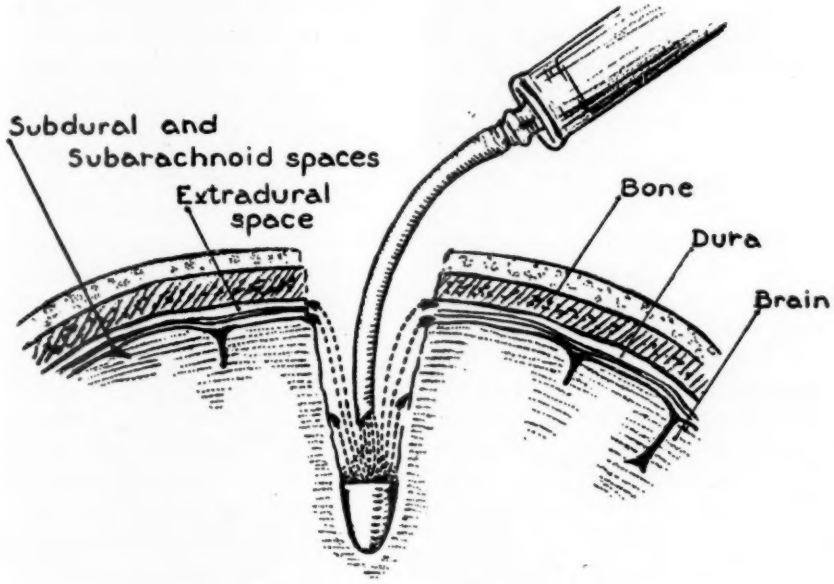

FIGURE 1. *Diagrammatic Representation of the Irrigation of a Bullet Tract in the Brain.*

In the skull, however, the circumstances are radically different, since any compound fracture necessarily communicates with one or more of the intracranial meningeal spaces. These are only locally contaminated at the time of the infliction of the wound. Irrigation of such a wound inevitably leads to the deposition of some of the irrigating fluid—loaded with débris and contaminating organisms—in hitherto uninvolved and uncontaminated meningeal spaces (Fig. 1). The greater the amount and the stronger the flow of the irrigating fluid, the wider the contamination. That this is a real and not a theoretical danger is recognized even by the advocates of irrigation. They one and all advise that the irrigation be gentle. No matter how gentle it is, however, the fluid of necessity seeps into areas that would not have become involved had no fluid been used.

It is also argued that in these wounds—especially at the time of operation—the meningeal spaces are mechanically closed by the swollen brain. This may be so macroscopically, although watertight closure is extremely unlikely under such circumstances, but it is certainly not so microscopically. Since the spread of contamination is by

micro-organisms, macroscopic closure is of little importance. Furthermore, because the danger of irrigation lies in the flow of fluid and the benefits in the outgoing current, any procedure that does away with the former while retaining the latter is not only an acceptable substitute but is preferable. Such a procedure is found in suction. Here the movement of the débris is entirely outward; its removal is positive and under the constant and direct control of the operator, and is capable of being carried out through the medium of various-sized suction tips that may, if necessary, be either smaller than the smallest practical rubber catheter or as large as the occasion demands. A variation in the strength of the suction governs the amount of tissue that can be removed. By reducing the intracranial pressure by means of lumbar cerebrospinal-fluid drainage during operation, as suggested by Cone,[11] the edges of the brain wound separate and gape. Exposure is improved, bleeding vessels can be held in the sucker mouth until closed, and the procedure, being technically simple, is more effective than irrigation. Certainly there can never be a reason sufficient to justify irrigation of a compound fracture of the skull previous to complete débridement, and in my hands and on theoretical grounds it has been and is not only unnecessarily dangerous but also inefficient, when compared to suction alone at the time of the complete débridement.

The other important cause of unnecessary spread of contamination beyond the wound is found in the method of applying the first dressing. It is not necessary to re-emphasize the dangers that go with manipulation and the advantages that accrue to the patient through lack of handling of the wound. It has been my custom to limit the first dressing to the application of one or more sterile gauze pads to the wound. These are held in place with a bandage. Until such time as the patient is anesthetized and about to be subjected to a major débridement, all such procedures as cutting the hair, shaving the scalp, washing or wiping the wound, removal of foreign bodies or fragments of bone, ligature of all except the superficial temporal and great occipital arteries, and retraction of the wound edges have been rigorously avoided.

With the advent of the sulfonamides and the recognition of their great value as bacteriostatic agents when powdered into a wound, I shall in the future permit sufficient cutting of the hair and wiping of the surface of the wound to allow for a reasonably efficient application of the powdered form of one of this group of drugs to and around the wound at the first dressing. Shaving of the scalp and washing of the wound at the first dressing, however, even when combined with chemotherapy locally, as advocated by Cloward,[12] are in my opinion contrary to all logic and cannot be too strongly condemned. In the necessary absence of figures, the good results that Cloward reports must be looked on as having occurred in spite of rather than because of this first aid.

It was intended that this entire series of 218 patients with compound fractures of the skull should be treated according to one technic.[13, 14] All the patients were supposed to have been operated on as soon as their general condition would permit it to be done safely, and in any event within forty-eight hours of the infliction of the injury. As a result, the majority of operations were performed within twenty-four to thirty-six hours. The first dressing was supposed to have been as described above. The débridement was done in the operating room with the patient usually under a general anesthetic, such as ether by inhalation or Pentothal intravenously. As a rule, local anesthesia was not suitable because of the type of patients that makes up my clinic. The whole head was clipped and shaved, scrubbed for ten minutes with soap and water, washed with alcohol and then ether, and the scalp painted with a 0.2 per cent alcoholic solution of iodine after the patient had been placed on the operating table. The wound, which had been closed by sterile gauze during this preparation, was then cultured, often from several levels, after which it was thoroughly swabbed inside with 7 per cent tincture of iodine. Two teams of assistants were used. One team helped the surgeon do the débridement. The other — usually made up of two men — inserted a cannula into an ankle vein. This was kept open by dripping through it a 5 per cent solution of glucose in physiologic saline solution. Intravenous medication or an anesthetic was given when necessary by puncture of the rubber tubing of this apparatus. This team also collected blood for transfusion from donors and, by substituting this blood or plasma for the glucose solution, took care of any emergencies, such as surgical shock or hemorrhage, that arose in the course of the débridement.

The line of the incisions to be used was then scratched on the scalp, drapes were put in place, and the scalp and periosteal wound edges were excised *en bloc*, the instruments that were used in this excision being discarded. The bone was drilled in what was thought to be an uncontaminated area adjacent to the fracture line and the contaminated part of the fracture was cut loose, if possible in one piece, with the aid of a De Vilbiss rongeur. If the dura was intact and there was no reason to suspect the presence of a subdural

hematoma, it was not touched. If such a clot was suspected, however, the dura was opened without hesitation and the subdural space explored. Extradural hematomas were removed, and the source of the bleeding identified and closed. Tears in the venous sinuses were closed with muscle stamp grafts. If the dura was torn, its torn edges were excised, the tear being extended when necessary to expose properly the underlying cortex. Cortical lacerations and hematomas were removed by suction until all macerated brain tissue had been excised. Bullet tracts were cleaned of bone fragments as well as débris and followed down to the bullet, the latter being removed. In 1 patient, the bullet was found in the lateral ventricle. Intracortical hemostasis was attained by silver clips, the use of the coagulating current or muscle stamp grafts. No irrigation of any kind was permitted at any time. The dura was then closed over the cortical scar, making a relaxing incision laterally if necessary. Periosteum was used as a dural graft a few times, but fascia lata was not. The removed bone even if of large size was not replaced.

The scalp was closed tightly with two layers of interrupted silk stitches — one in the galea and one in the skin. The skin was scarified adjacent to all skin sutures deeply enough to draw blood, particular attention being paid to the corners. Mattress sutures of silkworm gut were occasionally used as tension sutures. All suturing was done from the periphery toward the center of the wound, and plastic types of scalp closure were used whenever necessary. No drain or pack was used in simple compound fractures.

Compound fractures involving the paranasal sinuses were prepared and débrided similarly but were drained by a Mosher copper-wire cone drain, iodoform gauze and boric-ointment gauze strips. Patients with cerebrospinal fluid rhinorrhea were operated on by way of a Frazier type of right frontal flap, which has been described elsewhere.[14]

As a means of measuring the efficacy of this technic, I have eliminated from this series all those patients whose death or postoperative sepsis could be directly traced to errors in carrying it out. For the same reason, the patients who died before being operated on have also been eliminated.

As already stated, there were 60 deaths (Table 2). The causes were as follows: surgical shock, 28 cases; circulatory failure, 5; the cerebral condition, 6; meningitis, 13; cortical abscess, 3; pneumonia, 1; gas-bacillus infection of the leg, 1; acute edema of the glottis, 1; transection of the cervical cord, 1; and an unknown cause, 1. Of these deaths, 1 from circulatory failure, 3 from cortical abscess and 6 from meningitis can be eliminated because of gross technical errors of therapy. Thus, the deaths can be reduced by 10 to 50 and the mortality from 28 to 23 per cent.

As stated above, 159 patients were operated on and 25 of these died. Thirty-nine cases (25 per cent) were classed as septic following operation. If 29 patients whose death or sepsis was traced to technical errors are eliminated, 130 patients may be considered as having been properly operated on. Twelve of these died, reducing the operative mortality from 15.7 to 9.2 per cent. Ten patients (7.6 per cent) developed postoperative sepsis that could not be accounted for by any error in technic. Thus, this mortality rate (9.2 per cent) and this rate of postoperative sepsis (7.6 per cent) must be considered as being inherent in the operative technic as outlined above. These figures must be improved by the use of chemotherapy if the latter procedure is to justify itself.

There can be no question of the value of any of the sulfonamide drugs as bacteriostatic agents when powdered into the usual wound, either with or without additional oral administration. Moreover, there is no evidence that, when these drugs are used intelligently and in reasonable amounts, any harm comes to the tissues locally or to the patient in general. It has been assumed that what holds true for usual wounds also holds true for craniocerebral wounds. Recent work, however, suggests that this conclusion is not justified and that unexpected and unpleasant side effects may follow the use of certain of the compounds, and that care must be exercised in the amount used of all of them. Hurteau,[15] working with cats, demonstrated that following local application to a cerebral wound sulfanilamide was the most rapidly absorbed, sulfathiazole second, sulfadiazine third, and sulfapyridine the least rapidly absorbed. Furthermore, sulfadiazine exercised no untoward effect on the final result of wound healing and caused only negligible foreign-body reaction in the meninges. Botterell, Carmichael and Cone[16] also worked with cats and applied the sulfonamide drugs to the cut surfaces of a cerebral wound. Their experiments indicated that the intensity and duration of the inflammatory reaction depended on the quantity of the drug used, its solubility and the length of time it remained in the wound. They also observed no undesirable side effects on nerve cells, neuroglia or myelin at a distance from the drugs and stated that the reaction remained local. As a result of their experiments they have not hesitated to use sulfonamides in wounds of the human brain but have used them circumspectly. They further state that they will continue to use them guardedly even when their local bac-

teriostatic and bacteriolytic action is proved beyond doubt, "for they do act also as foreign bodies." The implantation of the drugs in the ventricle did not alter their findings.

More recently Watt and Alexander[17] report epilepsy following the use of sulfathiazole in the wounds of 5 patients on whom frontal craniotomies were performed. The drug was in contact only with the surface of the cerebrum. As a result of this experience, they applied the compound on the surface of the cerebrum in 10 cats. Nine developed epilepsy. Sulfadiazine (in 2 cats) and sulfacetamide (in 3 cats) were used in a similar fashion without producing any cerebral symptoms. It is to be noted that in both their clinical cases and experimental animals it was sulfathiazole that produced the epilepsy, and that furthermore, in contradistinction to the experimental work reported above, the drug was applied to the intact cerebral surface and not to the cut edges of a subcortical laceration. Pilcher et al.[18] have had similar experiences in experimental dogs and a few cats with this same drug, and have also noted the increased toxic effect when the drug was applied to the unbroken cerebral surface as opposed to the cut surfaces. Ingraham and Alexander[19] state that sulfadiazine powder can be safely used when the brain is not severely damaged, but suggest that further work must be done to arrive at a definite conclusion.

In 1 of my cases included herewith, sulfanilamide powder was used in the wound and sulfapyridine by mouth. No surgery was performed. The wound granulated, and when explored at the end of two weeks because of failure to heal was found to contain a "rock" of sulfanilamide. This was removed, after which the wound healed promptly.

In so far as the available evidence indicates, then, sulfathiazole should not be used in craniocerebral wounds. Sulfanilamide may be expected to give a high concentration of the drug for a short time, and sulfadiazine a lower concentration for a longer time. The evidence concerning the toxicity of sulfapyridine in craniocerebral wounds is inconclusive, and as a result, this drug should not yet be employed in such wounds. Care should be taken to use only the amounts of powdered crystals that are necessary, because it is evident that the powder acts as a foreign body when used in craniocerebral wounds, and may actually impede healing through creating a foreign-body reaction, in spite of the absence of any bacterial contamination.

Efforts directed toward lowering the mortality and the percentage of postoperative sepsis arising out of compound fractures of the skull through the use of sulfonamide products should be concentrated on the use of either powdered sulfanilamide or sulfadiazine crystals, applied in and around the wound at the first dressing and at the débridement. Sulfathiazole should never be used, and sulfapyridine only in the absence of any of the other forms. The crystals should be powdered and applied to the wound surface by an insufflator. No more than a thin layer of the drug should be applied. This chemotherapy is not and cannot be a substitute for properly conceived surgery in the treatment of compound fractures of the skull. It is an adjunct to such surgery, valuable without doubt but entirely incapable of replacing the latter.

Summary

An analysis of 218 cases of all types of compound fractures of the skull is presented from the point of view of complicating major injuries, age and gross, operative and nonoperative mortality.

Four principles are described as fundamental in the surgical therapy of compound fractures of the skull. They are as follows: All such wounds must be completely débrided within forty-eight hours of the time of infliction of the injury or else not operated on until completely healed for six to eight months. No patient should be operated on until he is out of surgical shock and until his general condition warrants it. After the diagnosis has been made by palpation through the wound, the first and only dressing prior to débridement must be one that can be applied with an absolute minimum of handling of the wound. Lastly, the débridement should be complete except in certain special fractures, it must be done in such a way as to avoid the spreading of bacterial contamination throughout the wound and the production of tissue necrosis, it should include the removal of all large foreign bodies, and no wound that has been properly débrided either needs to or should be drained.

A detailed discussion of the technic of the débridement is given. Irrigation of the wound before and during operation is condemned and the reasons therefor are given.

Chemotherapy in the form of sulfanilamide or sulfadiazine is recommended both by mouth and in the wound, but only as an adjunct to properly conceived and executed surgery. Sulfathiazole should not be used in craniocerebral wounds.

With therapy based on the above principles and without the use of chemotherapy, and with débridement done according to the above technic, the mortality in 130 patients who had been prop-

erly operated on was 9.2 per cent and the rate of postoperative sepsis was 7.6 per cent.

References

1. Miles, A. A., Spooner, E. T. C., and van den Ende, M. Discussion on infection of wounds. *Proc. Roy. Soc. Med.* 34:99-108, 1940.
2. Spooner, E. T. C. Bacteriology of air-raid wounds examined within forty-eight hours of infliction. *Brit. M. J.* 2:477, 1941.
3. Hare, R., and Willits, R. E. Bacteriology of recently inflicted wounds with special reference to haemolytic streptococci and staphylococci. *Canad. M. A. J.* 46:23-30, 1942.
4. Smith, A. N. Incidence of potentially pathogenic staphylococci in nose and on skin of healthy subjects. *J. Roy. Army M. Corps* 76:341-344, 1941.
5. Trueta, J. Discussion on infection of wounds. *Proc. Roy. Soc. Med.* 34:107, 1941.
6. Barnes, J. M. Spread of bacteria and toxins from infected wounds: clinical application of laboratory findings. *Proc. Roy. Soc. Med.* 34:659, 1941.
7. Cairns, H. Gunshot wounds of head in 1940. *J. Roy. Army M. Corps* 76:12-22, 1941.
8. Jentzer, A. Traumatismes craniens et plaies cranio-cérébrales par projectiles. *Schweiz. med. Wchnschr.* 21:648-667, 1940.
9. Tönnis, W. Die Behandlung der Hirnverletzungen auf Grund der Erfahrungen im Feldzug gegen Polen. *Deutsche med. Wchnschr.* 66:57, 1940.
10. Munro, D. Bacteriology of wounds of compound fractures of skull and its clinical significance. *New Eng. J. Med.* 227:939-944, 1942.
11. Cone, W. V. Personal communication.
12. Cloward, R. B. War injuries to head: treatment of penetrating wounds. *J. A. M. A.* 118:267-270, 1942.
13. Munro, D. Treatment of compound fracture of skull: study of 185 cases. *New Eng. J. Med.* 213:551-559, 1935.
14. *Idem. Cranio-Cerebral Injuries: Their diagnosis and treatment.* 412 pp. New York: Oxford Medical Press, 1938.
15. Hurteau, E. F. Intracranial use of sulfadiazine: experimental study of histology and rate of absorption. *Canad. M. A. J.* 46:15-17, 1942.
16. Botterell, E. H., Carmichael, E. A., and Cone, W. V. Sulphanilamide and sulphapyridine in experimental cerebral wounds. *J. Neurol. & Psychiat.* 4:163-174, 1941.
17. Watt, A. C., and Alexander, G. L. Epilepsy following application of sulphathiazole near brain. *Lancet* 1:493-495, 1942.
18. Pilcher, C., Angelucci, R., and Meacham, W. F. Convulsions produced by intracranial implantation of sulfathiazole. *J. A. M. A.* 119:927, 1942.
19. Ingraham, F. D., and Alexander, E. Experimental application of sulfonamide drugs to the cerebral cortex. *New Eng. J. Med.* 227:374-378, 1942.

SPINA BIFIDA AND CRANIUM BIFIDUM*

III. Occult Spinal Disorders

FRANC D. INGRAHAM, M.D.,† AND JOHN J. LOWREY, M.D.‡

BOSTON

ROUGHLY 25 per cent of normal children have occult defects in the vertebral laminas and this incomplete closure is frequently demonstrated in x-ray films taken for some other reason. The defect is likeliest to occur in the lumbar or sacral region and can be shown to have persisted in many normal adults. Over a twelve-year period at the Children's Hospital we have selected the much smaller group of patients who have had symptoms associated with occult spina bifida. Probably too few of these patients have been given the benefit of local exploration, but it is only by careful study and selection that operation can be offered to the proper group.

The variations of symptoms and physical findings are unlimited. In some cases there may be such faulty development of the lower portion of the spine and cauda equina that surgical treatment will not be helpful, and those patients who might possibly be worse after operation must be carefully excluded.

Presenting Symptoms and Signs

Of the 65 cases studied, 31 were first seen in the orthopedic clinic and the most frequent presenting complaint was maldevelopment of the feet or lower extremities with some disturbance in gait, which was present in 29 cases. The findings included valgus, varus and cavus deformities of a foot, a small foot and a limp due to muscle weakness. In the large majority of cases the deformity was unilateral. The second commonest complaint was some local abnormality, such as a swelling, a bony lump or defect, a scoliosis or a local overgrowth of hair. These accounted for 22 cases. Enuresis or incontinence brought 7 patients to the hospital. In 9 cases the spina bifida was an incidental finding.

The neurologic disabilities are not easily classified because of the difficulty of deciding whether or not a given abnormality is on a neurogenic basis. If a patient on physical examination shows anesthesia over the distribution of the fifth lumbar root and at operation is found to have a tumor pressing on that root, the cause and effect are apparent. In most of our cases, however, the findings at physical examination were various degrees of muscular weakness in a lower extremity and an abnormality of posture presumably dependent on this imbalance of muscle groups. The findings at operation suggest that the muscle weakness and imbalance may be on a neurogenic basis. The age group dealt with made neurologic examination difficult, but abnormal reflexes were recorded in 15 cases, a positive Babinski response in 7, poor sphincter tone in 6, and abnormal sensation in the lower extremities in 5.

Besides the neurologic or developmental changes in the lower extremities, which may or may not have been produced by lesions of the cord or

*From the Department of Surgery of the Children's Hospital, Boston, and the Harvard Medical School.

†Neurosurgeon, Children's Hospital; associate in neurosurgery, Peter Bent Brigham Hospital; associate in surgery, Harvard Medical School.

‡Formerly, fellow in neurosurgery, Children's Hospital, and assistant in surgery, Harvard Medical School.

cauda equina at the site of the spina bifida, there were a few local findings of importance. As determined by physical examination or x-ray or both, the lesion was present most frequently in the lumbosacral region (25 cases). Eighteen cases showed sacral involvement alone, 4 dorsal, 3 dorsolumbar and sacral, 3 dorsolumbar, 1 cervicodorsal, and 1 cervical.

As stated above, some local abnormality, such as a soft or bony swelling, a local overgrowth of hair or a palpable defect, was the presenting complaint in 22 cases. Routine physical examination showed a palpable bony defect in 28 cases, and a local overgrowth of hair in 14. Of these 65 cases, in 13 the abnormality was associated with a so-called "lipoma." As described in a previous paper,[1] lobules of gritty, firm fat, bound together by fibrous septa, usually adherent to adjacent fascial (or dural) planes by firm and often vascular attachments are frequently found associated with spina bifida. They may be at any tissue level from the cord itself to the subcutaneous tissue or may extend directly from one layer to another, binding the various tissues together and thereby producing adhesions and preventing the normal movement of the caudal end of the spinal cord with growth.

It should be emphasized, however, that spina bifida may be present without any symptoms or signs. It was present in 11 cases without any neurologic disability or abnormality of the lower extremities, such as valgus or small foot, that might cause a disturbance in gait. In 15 cases, there was no local visible or palpable abnormality that would suggest the spinal defect.

Operated Cases

Of the 65 cases reported, 26 had exploration of the region of the spina bifida. In 4 of these, the procedure was minor in nature and consisted only in local excision of a subcutaneous lipoma or dermoid, or incision and drainage of an infected dermoid. In 22 cases, a laminectomy was performed. One of these was in a five-day-old infant with bilateral brachial palsy. At operation acute angulation of the cord due to hemivertebras was found. The patient died two days after operation. Eliminating this case as being unusual and complicated by the other vertebral anomalies in addition to the spina bifida, there were 21 cases. Some of these patients had two-stage operations. Many of them had previous or subsequent orthopedic operations to correct the deformities of the extremities.

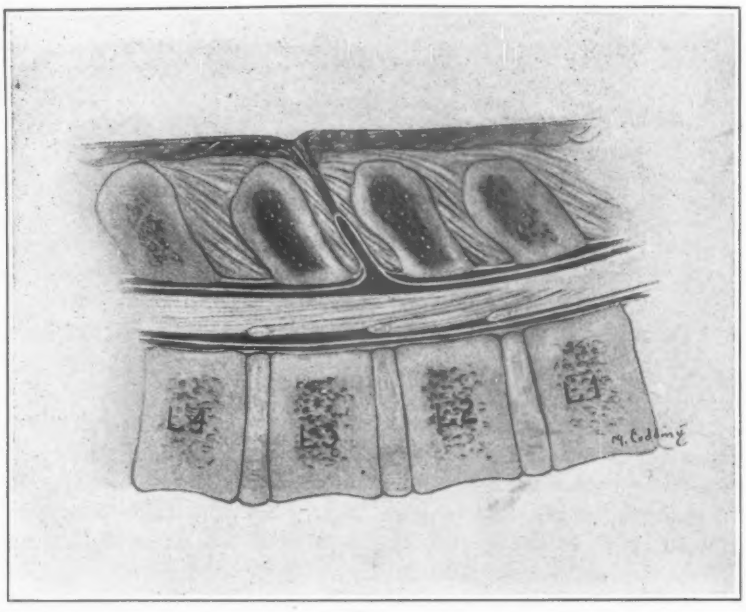

Figure 1. *Fibrous Stalk Connecting Dimpled Area in Skin with a Protrusion of the Dura between Laminas.*

The findings at operation were variable. The most frequent finding was a combined intradural and extradural lipoma, which was present in 5 cases. Three cases showed extradural lipoma, 3 a fibrous tract leading from the skin to the dura, 2 subdural lipoma, 2 extradural lipoma with a bifid spinal cord, 2 no gross lesion of the spinal cord or the nerve roots, 1 an abnormal spicule of bone with pressure on the nerve roots, 1 a fibrous tract from the skin to the ligamentum flavum,

1 a constriction of the cord due to adhesions, and 1 a meningocele entirely contained within the sacral canal.

The criteria on which success of operations of this sort must be based are difficult to determine because of the multiple complaints. Also, where permanent nerve damage has been present it is not possible to expect improvement. It therefore seems justifiable to consider a result as good if a definite cause of progressive symptoms can be found and relieved and if no further progression of symptoms occurs.

There was no operative mortality in these cases. In some cases there was striking recovery, such as healing of a trophic ulcer with return of sensation, or marked improvement in urinary and fecal control. It is important that patients who are developing equinus or cavus deformities be operated on early, because such deformities once present cannot be expected to disappear following operation. Using the criteria defined above, it can be said that a good result was obtained from operation in 13 cases. In 3 cases the cord or roots showed an abnormality and the disturbance in gait persisted. Three cases have not been followed long enough to determine the result, although all these showed obvious disease at operation. One showed a bifid spinal cord. One patient (Fig. 1) operated on because of incontinence, with the finding of a fibrous cord from the subcutaneous tissue to the dura, exerting tension on the latter, showed improvement in the hospital but lapsed to practically her preoperative state when left with unco-operative parents. One patient awaits reoperation because complete removal of an extradural lipoma was not possible at the first operation.

FIGURE 2. *Case 2.*

The photograph on the left shows the abnormal prominence of a spinous process and dimpling of the skin over the second lumbar vertebra; that on the right, the trophic ulcer of the toe.

CASE REPORTS

CASE 1 (C. H. 75872). This patient was first seen in the hospital at 2 months of age because of bilateral club feet. The feet were treated with manipulation and then casts. At the age of 4 he was admitted because of a trophic ulcer of the right small toe (Fig. 2). This failed to heal and the toe was amputated. Spina bifida occulta

was noted at that time. He was followed in the Out Patient Department and did fairly well except for occasional trophic ulcers. At the age of 8 he had a right arthrodesis for persistent varus deformity of the right foot. At that admission reflexes in the right leg were absent and there was diminished sensation over the lower half of the lateral surface of the lower leg and the dorsum and sole of the foot. At 11 years of age the patient was admitted for a trophic ulcer of the right fourth toe, which subsided following treatment with wet packs.

Four months later he was admitted for a trophic ulcer of the right third toe that had developed cellulitis.

Physical examination showed a large crater over the third toe discharging purulent material, with surrounding cellulitis. The diminished sensation over the lower leg and foot was still present, with absent temperature sense. The spine showed a dorsal angulation over the 2nd lumbar vertebra with a palpable bony defect. X-ray examination showed abnormal development of the 12th dorsal vertebra and all the lumbar and sacral laminas, with incompletely formed, defective and incompletely fused arches. It was decided that there might be pressure on the spinal cord associated with the spina bifida, so laminectomy was carried out. On opening the dura definite constriction of the cord due to adhesions associated with the defective closure of the spinal cord was found at the level of the 11th dorsal vertebra. The cord was freed at that point.

Postoperative recovery was uneventful. A small piece of bone was removed from the base of the ulcer 7 days postoperatively, following which healing rapidly occurred. Postoperatively there was return of sensation and improvement in control of the anal sphincter. When the patient was last seen, 10 months after operation, his toes were still in good condition.

Case 2 (C. H. 263163). This girl first entered the hospital on the Neurological Service at 9 years of age with a chief complaint of progressive talipes equinovarus deformity of both feet of 4 years' duration. The feet tended to become cold. Otherwise there were no complaints. The patient had had treatment with corrective shoes and arch supports without relief. The family and past histories were not remarkable.

Physical examination showed diffuse mottling of both lower extremities, with atrophy of the legs from the knees down, talipes equinovarus deformity of the feet and thick calluses on the toes. Neurologic examination was negative except for diminution of pain and touch sensation in the toes bilaterally. X-ray films of the spine showed a spina bifida of the 3rd lumbar vertebra and of the sacral segments, with a poorly formed sacrum.

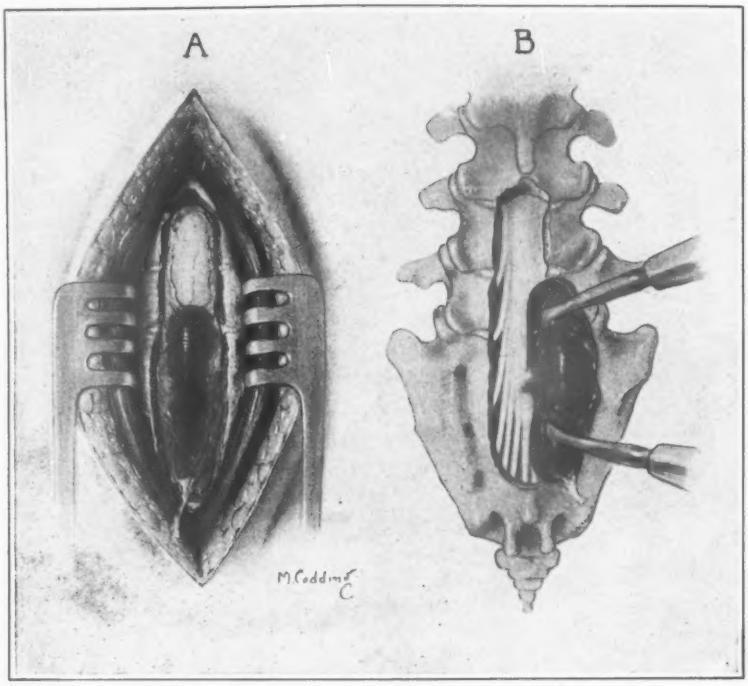

Figure 3. *Case 2.*
These drawings show the intraspinal meningocele connected with the dura by a small stalk.

Laminectomy was performed in two stages owing to the extensive dissection necessary, and a meningocele (Fig. 3) was found extending from the lumbosacral junction through the sacral canal to the undersurface of a dimple in the skin just above the anus. The sac contained dark-yellow fluid. No connection with the dura proper could be demonstrated except for a tiny fibrous stalk. The sac was freed from the nerve roots and excised.

Postoperatively the patient did well. Six months after operation she showed return of sensation to the toes, and the temperature of the lower legs seemed normal. She walked better than before the operation. Muscle examination showed essentially the same picture as preoperatively.

Case 3 (C. H. 244120). This girl was first seen in the Orthopedic Clinic at 9 years of age for a deformed left foot, which had been present at birth.

Physical examination showed atrophy of the left lower leg and equinovarus deformity of the foot, associated with a cock-up of the toes (Fig. 4). The left foot was considerably smaller than the right and there was some diminished sensation over it. The spinous process of the 3rd lumbar vertebra appeared double. X-ray examination showed congenital anomalies of the laminas of the 2nd and 3rd lumbar vertebras, with partial fusion of the laminas and indentation of the posterior aspects of the vertebral bodies. There were also defects in the laminas of the 5th lumbar vertebra and the 1st sacral segment.

Lumbar puncture was done, with normal spinal-fluid findings, and the patient was discharged home. She was followed in the Out Patient Department over the next year and developed further loss of sensation in the left foot and also loss of reflexes on the left. Lumbar laminectomy was therefore carried out. A lipoid mass was found in the subcutaneous tissue, and extending from this was a fibrous stalk that penetrated the edge of the 3rd lumbar vertebra and entered the neural canal, but without any connection with the dura (Fig. 5). On opening the dura, small swellings giving the appearance of neuromas, the maximum being twice the diameter of the nerve trunk, were seen on the 2nd, 3rd and 4th lumbar roots. These swellings could not be isolated and no attempt was made to resect them. The canal was left wide open.

The patient had a smooth postoperative course and was discharged home on the 16th postoperative day immobilized in a plaster jacket. At the time of discharge the area of hypoesthesia had diminished so that instead of half the dorsum of the left foot only the toes of the left foot were affected. Muscle examinations before operation and 5 months postoperatively showed no change in muscle strength.

FIGURE 4. *Case 3.*

The photograph on the left shows the short left leg and atrophy of the muscles of the calf; that on the right, the deformity of the left foot, as seen from below.

CASE 4 (C. H. 239250). This girl was first seen in the Orthopedic Clinic at 5 years of age, with a complaint of weakness of the left foot. Five months previously it had first been noted that the left foot was smaller than the right and that the patient seemed to drag it while walking. These complaints had progressed to entry.

Physical examination was normal except for the spine and lower extremities. There was a growth of long, dark hair over the lower lumbar and upper sacral segments, with a suggestive underlying spinal defect (Fig. 6). The left foot was smaller than the right and showed a cavus deformity. The left calf was 0.5 cm. and the left thigh 1.0 cm. smaller than the right. Reflexes and sensation were normal in both lower extremities. X-ray films showed defects involving the laminas of the first three lumbar and upper sacral segments, with widening of the interpeduncular spaces. The spinal fluid was normal.

At laminectomy it was found that the spinous processes of the 3rd and 4th lumbar vertebras were absent and

that the laminas were overlapped, those on the right lying under the left so as to cause constriction of the dura and absence of epidural fat. The dura was exposed and opened, and chronic arachnoiditis was found. On the left, in the dura at the level of the 4th interspace, there was a funnel-shaped depression that was attached to a piece of bone coming from the ventral surface. The nerve roots on the left were deflected by this bone and were adherent to it. The nerve roots were freed, and the bony prominence removed.

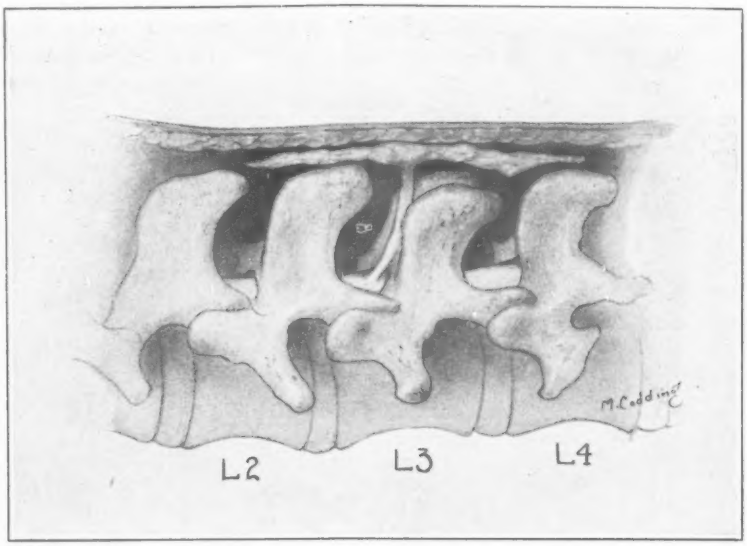

FIGURE 5. *Case 3.*
This drawing is a reconstruction of the firm lipoma extending from the dura to the subcutaneous tissue.

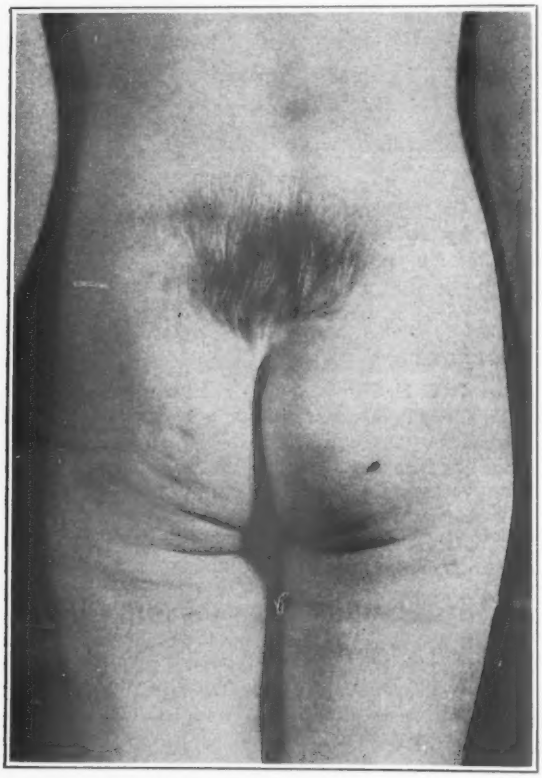

FIGURE 6. *Case 4.*
This photograph shows the abnormal growth of hair over the site of the spina bifida.

Postoperatively the patient made a good recovery and was discharged in a plaster jacket on the 14th postoperative day. With orthopedic shoes she did well. The gait was better and the deformity did not progress.

SUMMARY

An analysis of 65 cases of spina bifida occulta is presented. The symptoms and usual findings are listed.

Twenty-one cases in which laminectomy was performed are discussed, and the operative findings summarized.

The indications for operation were specific, and operation was not performed without a definite history of progressive difficulty.

In only 2 out of the 21 cases did operation fail to show a lesion that might account for the presenting complaint. In the large majority operation showed a definite lesion affecting the cauda equina or nerve roots, that could be remedied surgically.

The findings suggest that probably more of the questionable cases should be offered the benefit of exploration.

REFERENCE

1. Ingraham, F. D., and Swan, H. Spina bifida and cranium bifidum. I. Survey of five hundred and forty-six cases. *New Eng. J. Med.* 228:559-563, 1943.

RESECTION OF THE BLADDER NECK FOR OBSTRUCTION IN WOMEN

Report of a Case

CHARLES MIRABILE, M.D.*

HARTFORD, CONNECTICUT

OBSTRUCTION of the vesical neck in females as compared with that in males occurs infrequently. Young,[1] who reviewed the literature in 1938, was able to find only 36 cases in females. The condition is undoubtedly commoner than this figure suggests, and the smallness of the figure must be attributed to lack of recognition of cases on the one hand, and failure to report recognized cases on the other. In any event, obstruction in females is a rarer event than in males, and the basic cause is to be found in the simpler structure of the female urethra (Lewis[2]). This simpler structure does not predispose the female urethra to the frequency and to the degree of inflammatory lesions that may lead to sclerotic changes at the bladder neck.

That the female urethra is the embryologic homologue of the portion of the male urethra that lies between the vesical orifice and the prostatic utricle was well shown by Johnson[3] in 1922. By means of wax reproductions of the urethras of female embryos, he demonstrated that the female urethra contained branching urethral glands that he believed to be homologous to the male prostate. These glands may become infected and result in a condition equivalent to the chronic prostatitis observed in the male.

Young,[4] in his recent book on genital abnormalities, has reported a case in which the female urethral glands had developed in size to a point approaching that of the male prostate. There was a hyperplasia of the adrenal cortex, giving rise to a masculinizing effect due to the increase of androgenic substance. The female urethra may therefore be the seat of the same type of disease as that observed in the male. Thus, there can be chronic infection of the glands, — corresponding to chronic prostatitis, — fibrosis with a median bar or contracture of the neck of the bladder, and finally, in cases of glandular dyscrasia, adenoma of the male can be simulated. Furthermore, these conditions probably arise from the same causes as they do in the male, whatever those causes may be.

Caulk,[5] in 1921, first called attention to bladder-neck obstruction in females, which he treated successfully by means of his punch. He believed that the obstruction was due to fibrosis and hyperplasia at the bladder neck resulting from three causes: inflammation; trauma at childbirth and trauma from sexual activity.

Young[1] suggested that when the female urethra became infected, the urethral glands caused infection to persist, subsequently leading to fibrosis. Thompson,[6] who reported 24 cases of vesical dysfunction in females, agreed that chronic inflammation was the most important cause, but added that in some cases there was an additional factor in the form of nervous imbalance between the sphincter (parasympathetic innervation) and the detrusor muscle (sympathetic innervation), which led to spasm of the sphincter and subsequent hypertrophy and hyperplasia of the muscle.

In considering the etiology of bladder-neck obstruction in females, the common denominator appears to be chronic inflammation in the form of a nonpurulent urethritis.

The symptoms of obstruction in females are precisely the same as those observed in males, namely, hesitation of the stream, difficulty in starting it, its slowness and so on, leading to complete retention. The cystoscopic picture, like the symptoms, is strikingly similar in both sexes, with marked trabeculation and sacculation, and in advanced cases, the accumulation of residual urine. The cystoscopic picture at the bladder neck, however, may be quite unrevealing, showing only a minimal amount of tissue in the form of a ridge or bar, which, taken alone, is not sufficient for diagnosis. In making the diagnosis it is necessary to take into account the secondary signs of obstruction, which are to be found in the bladder and in the upper urinary tract and which take the form of dilatation due to back pressure. The presence of residual urine is important.

In the treatment of this condition, as the following case well illustrates, the passage of sounds, even those of large caliber, is not sufficient to control the situation. Where the lesion is a localized stricture of the urethra, the passage of sounds constitutes adequate treatment, as it does in the male. To quote Kelly,[7] "If there is much infiltration above the localized stricture area making restoration of the tissues a hopeless task, one may then make an annular resection of the urethra, cutting out the diseased parts." Kelly thus recognized that certain types of obstruction of the

*Associate urologist, Hartford Hospital.

female bladder neck do not respond to simple dilatation. The case to be presented demonstrates this very well, for previous to operation, sounds were passed to little or no effect. The treatment of this condition in females, therefore, as in males, is transurethral resection, and rarely does one ever have to do so little to achieve so much. The making of several incisions with the removal of the corresponding number of pieces of tissue is all that is necessary. Because the female urethra lies on the superior vaginal wall throughout its course, great care must be taken to avoid cutting too deeply and thus causing the formation of a vesicovaginal fistula.

The matter of incontinence following resection of the bladder neck appears to be important only in theory, for in practice, incontinence was observed by none of the authors reporting punch and electrical-resection operations. Because obstruction in the female is accompanied by the same impairment of renal function as in the male, preoperative catheter drainage is as necessary in the former as in the latter.

Case Report

S. S., a 54-year-old, married woman, was seen in November, 1940, 4 days after an attack of acute retention. There was a history of difficult urination of 16 years' duration, culminating in acute retention. The patient had had two children, each labor not being unduly difficult so far as she could recall. There was nothing to suggest a previous infection of the urethra. She was first seen in the office, where the urethra was dilated by means of sounds (up to No. 28 Fr.) and a catheter was passed to the bladder. About 2000 cc. of cloudy urine was removed, although the patient had voided just before examination. A smear of the catheterized urine revealed many gram-negative bacilli, probably colon bacilli. The patient was advised to go to the hospital, but because the passage of sounds had given some relief she did not do so until January, 1941.

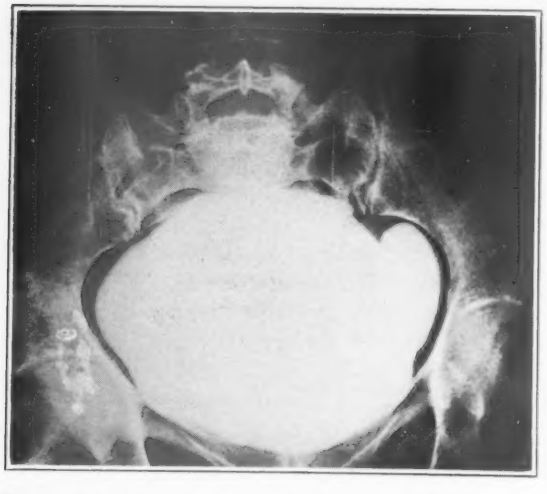

Figure 1. *Anteroposterior Cystogram Showing Enormous Dilatation of the Bladder and Two Large Diverticula.*

At that time cystoscopy revealed a markedly dilated bladder containing over 2000 cc. of cloudy urine. The bladder wall was trabeculated and sacculated, and just behind the trigone were the openings to two large diverticula. At the bladder neck there was a moderate ridge or bar which, however, did not appear particularly obstructive. A cystogram (Fig. 1) and a pyelogram (Fig. 2) confirmed the cystoscopic findings, and operation was decided on.

Figure 2. *Bilateral Pyelogram Showing Beginning Back-Pressure with Slight Dilatation of the Pelvis and Blunting of the Calyxes on the Right.*

The nonprotein nitrogen on admission, interestingly enough, was 36 mg. per 100 cc., suggesting that the bladder had acted as an excellent buffer, protecting the upper urinary tract from the back pressure. With a Foley bag catheter in place a phenolsulfonephthalein function test was done. It showed 19 per cent excretion in the first 15 minutes and 13 per cent in the next 45 minutes, or a total of 32 per cent in 1 hour. The volume of urine excreted in 1 hour was 750 cc.

At operation the diverticula were removed; inspection of the internal orifice showed this to be high in position, but it admitted the tip of the index finger fairly easily. It was decided that the diverticulectomy, followed by periodic passage of sounds, would serve to control the situation.

The postoperative course was uneventful, except that before the suprapubic sinus became completely dry, a retention catheter had to be inserted and left in place for 2 days. The patient was discharged from the hospital

to be followed in the office. Her residual on discharge was 100 cc., but over a period of 2 months it varied from 100 to 450 cc. and was usually around 300 cc. The urine was infected, and irrigations and the administration of sulfonamide drugs failed to clear up the infection. Accordingly, the patient was told that she had an obstruction at the bladder neck that refused to respond sufficiently to dilatation, and that it would be necessary for her to re-enter the hospital for operation.

FIGURE 3. *Photograph of Section of Tissue Showing Extensive Fibrosis and Absence of Glandular Elements.*

The patient did so in April, 1941, and transurethral resection was done. Six cuts were made, with the removal of no more than 2 gm. of tissue (Fig. 3). A retention catheter was inserted and left in place for 2 days, and when this was removed the patient voided promptly and freely and emptied the bladder completely. The urine became free of infection in 10 days.

The patient was examined in April, 1942, 1 year after transurethral resection. At that time the residual urine was nil and the urine was completely free of infection. A cystogram (Fig. 4) revealed that the bladder capacity was about 350 cc. and that the bladder function was perfect.

FIGURE 4. *Anteroposterior Cystogram after Operation.*

SUMMARY

The pathology, etiology, symptoms and treatment of obstruction of the bladder neck in women are briefly discussed, and a case is reported.

179 Allyn Street

REFERENCES

1. Young, H. H. Pathology and treatment of obstructions at vesical neck in women. *J. A. M. A.* **115**:2133-2136, 1940.
2. Lewis, E. C. Some aspects of urology in women. *Clin. J.* **66**:406-411, 1937.
3. Johnson, F. P. Homologue of prostate in female. *J. Urol.* **8**:13-34, 1922.
4. Young, H. H. *Genital Abnormalities, Hermaphroditism and Related Adrenal Diseases.* 649 pp. Baltimore: Williams & Wilkins Company, 1937.
5. Caulk, J. R. Contracture of vesical neck in female. *J. Urol.* **6**:341-343, 1921.
6. Thompson, G. J. Transurethral operations on women for relief of dysfunction of vesical neck. *J. Urol.* **41**:349-359, 1939.
7. Kelly, H. A., and Burnham, C. F. *Diseases of Kidneys, Ureters and Bladder: With special reference to diseases in women.* Vol. 2. 652 pp. New York and London: D. Appleton and Company, 1922. P. 586.

MEDICAL PROGRESS

EPIDEMIOLOGIC ASPECTS OF FOOD-BORNE DISEASE

VLADO A. GETTING, M.D., DR.P.H.*

BOSTON

A VAST variety of diseases may be transmitted through the medium of food, milk or water. Typhoid fever, paratyphoid fever, botulism and dysentery are classic examples of bacterial diseases that are transmitted to the unsuspecting consumer through the food and drink he ingests. A food-borne disease is an illness whose etiologic agent gains entrance to the body by the alimentary tract. In the past the term "food poisoning" has been applied conventionally to any acute gastroenteritis characterized by nausea, vomiting and diarrhea. However, a substantial portion of so-called "food poisoning" is not poisoning but an infection, and this term is therefore erroneous. "Ptomaine poisoning" is another misleading term often applied to an acute gastroenteritis. As a matter of fact, as is well stated by Whipple,[1] "ptomaine poisoning doesn't exist." Ptomaines are substances produced in the decomposition of food by bacteria. Examples of this type of decomposition are the ripening of game and wild fowl and the aging of cheese. None of these foods that are relatively high in ptomaines are poisonous.

CLASSIFICATION OF DISEASES

The food-borne diseases can be divided into two main groups: food infection, in which the etiologic agent is a living organism and the symptoms are the manifestation of the reaction of the host to its action; and food poisoning, in which the causative agent is a noxious substance that in itself is responsible for the characteristic clinical sequence, with or without the presence of a living organism.

The etiologic agent of food infection may be a virus, a bacteria, a protozoa or a helminth. By far the commonest agents are bacteria, chiefly the Salmonella and the Shigella. Trichinosis is perhaps the most prevalent type of helminth food infection, and amebiasis the commonest protozoan infection. An example of virus food-borne infection is infectious diarrhea of the newborn. The following food infections are reviewed in this paper: amebiasis, trichinosis, typhoid fever, paratyphoid fever, bacillary dysentery, undulant fever, scarlet fever and septic sore throat.

Food poisons may be subdivided into two groups: endogenous poisons, which are usually present in the food, — for example, mushroom poisoning or oxalate poisoning from rhubarb leaves, — and exogenous poisons, which are not an integral part of the food but an addition to it. The exogenous poison may be an organic substance, such as the alkaloid derived from a flagellate, which at certain times of the year infests mussels, the toxin of *Clostridium botulinum* or the commonest type of food poison, the enterotoxin of a hemolytic *Staphylococcus aureus*. Poisoning from this group is often termed food intoxication, since the symptoms are usually due to a toxin. On the other hand, the exogenous poison may be an inorganic compound such as the residue of an insecticide spray on fruits or vegetables, the accidental addition of fluorine to food or the solution of cadmium from replated metal containers. This type of food poisoning is often called chemical poisoning. The types of poisoning to be reviewed here are staphylococcal enterotoxic poisoning, botulism and chemical poisoning.

SYMPTOMATOLOGY

In food infections, the incubation period varies from six hours to two weeks, with characteristic averages for the different types of organism. The shortest periods are encountered with Salmonella infections, but usually the incubation period of a food infection is longer than that of food poisoning. Whereas fever is a common symptom of food-borne infections, it is not a frequent finding in staphylococcal enterotoxic poisoning. Nausea, vomiting, abdominal cramps or pain and diarrhea, leading to prostration, are the usual rapid sequence in severe staphylococcal poisoning; chills, sweats and toxemia are other frequent symptoms. The duration is usually a matter of hours, and recovery is rapid. In Salmonella infection recovery is a matter of days. In the investigation of any suspected food-borne outbreak an analysis of the symptoms will often give the epidemiologist a clue to the etiologic agent. The shortest incubation periods are observed in some types of chemical food poisoning, where symptoms may occur in ten

The articles in the medical-progress series of 1941 have been published in book form (*Medical Progress Annual*. Volume III. 678 pp. Springfield, Illinois: Charles C Thomas Company, 1942. $5.00).

*Commissioner of public health, Commonwealth of Massachusetts; assistant in preventive medicine and epidemiology, Harvard Medical School and Harvard School of Public Health; formerly, commissioner of public health, Worcester, Massachusetts.

minutes to several hours. The enterotoxin of *Staph. aureus* produces symptoms in two to six hours, whereas Salmonella infection incubation averages six to twenty-four hours. Nausea or vomiting is not likely to occur in food infections with long incubation periods.

Investigation of Food-Borne Outbreaks

Having obtained a record of the symptoms of all the patients in a given outbreak, the next most important step is to obtain a dietary history from each. Dack[2] presents a questionnaire that may be particularly useful to physicians interested in the compensation and legal aspects of food poisoning. As soon as the investigator finds which foods are common to all cases, steps should be taken to prevent further cases. Thereafter a study of the vehicle can be made, determining its source, handling, storage and preparation, in an endeavor to ascertain the mode whereby the food became contaminated. Lastly, the etiologic agent is determined by laboratory examination of the food and of the vomitus and feces of patients and food handlers. A complete investigation can reconstruct the epidemic, step by step, from its source to the time of the investigation.

Modes of Contamination of Food

Food-borne infection may be due to animal disease transmitted through milk or meat directly to the consumer. Trichinosis is a classic example of animal disease transmitted through uncooked pork. Brucella and certain Salmonella and streptococcal infections are examples of bacterial diseases of animals transmitted to man. Food may be contaminated by flies, rats or mice, or by food handlers who are active cases or carriers and who soil their hands with saliva, urine or feces, thereby introducing the organisms into the food they handle. Infection of food with staphylococci may occur from boils, infected wounds or sore throats of food handlers. The storage of food is an important factor in the morbidity rate: the longer the infected food is kept in a warm place, the higher the attack rate. Vegetables may be infected with amebas by fertilizing the soil with human excreta. Botulism is caused by undestroyed spores of *Clostridium botulinum* derived from the soil. Chemicals may gain entrance to foods. An insecticide spray may be found on fruits or vegetables; roach powder, which contains fluorine, may be added to the food by mistake for flour or milk powder; or a solution in an acid medium of cadmium from replated food containers may contaminate their contents. Often the mode of infection of the food cannot be determined. Many food-processing methods may be the means of introducing pathogenic organisms unless proper precautions are taken. Some of the outbreaks reviewed here illustrate the protean modes whereby food becomes the vehicle of food-borne disease.

Prevalence of Food-Borne Disease

The serious nature of gastroenteritis in military forces can be estimated from the incidence of this disease in the United States Army. During World War I, according to Dunham,[3] 87,774 cases of enterocolitis and diarrhea were admitted to hospitals, with 195 deaths — an annual case rate of 21.26 per 1000 men. Undoubtedly there were many more cases that did not require hospitalization. The average time lost from duty was eleven days, a total of almost a million days in hospitalized cases alone. In 1934 and 1935 the rates of hospital admissions were 21.95 and 21.14 per 1000, respectively.

The German literature is full of accounts of bacillary dysentery among the German troops in Poland.[4] Rodenwaldt[5] states, "Dysentery is the most dangerous of war diseases not only because of its physical effects, but also because it has such a powerful demoralizing effect." During World War I, according to Wurm,[6] there were 155,000 cases of dysentery in the German Army. During the invasion of Poland, in September, 1939, several outbreaks of bacillary dysentery occurred simultaneously. These outbreaks were due to different organisms and were therefore unconnected. The speed of the campaign forced troops to eat whatever they could lay their hands on, including a great deal of unripe fruit, and the heat made them drink water regardless of its purity. Strangely, there was no epidemic among the Polish prisoners or the civilian population. Many German troops had to be evacuated to the rear, where, because of unfavorable conditions and overcrowding, dysentery spread among the hospital personnel.

However, food-borne disease is prevalent in peacetime as well as wartime. In 1923, the United States Public Health Service began the collection of data on milk-borne disease.[7] Prior to this date, knowledge of milk-borne outbreaks was limited to those reported in the literature. As shown by these surveys, the number of known milk-borne outbreaks in the United States increased from an average of 17 per year prior to 1923 to an average of 42 per year since that date. From 1938 to 1941, inclusive (Table 1) there were 163 outbreaks, with 6923 cases and 48 deaths. In 1938,[9] there were 42 outbreaks, with 1587 cases and 27 deaths.[8] All these outbreaks were due to raw milk or raw-milk products, with the single exception of an outbreak traced to improperly pasteurized milk. In 1939, there were 41 outbreaks, with 2509 cases and 7

deaths.[9] Thirty-seven of these were traced to raw milk and 4 to milk that became contaminated subsequent to pasteurization. In 1940, there were 43 outbreaks, with 1658 cases and 10 deaths.[10] The largest of these was an epidemic of septic sore throat due to a hemolytic streptococcus at Dighton and Taunton, Massachusetts. Investigation revealed that members of the milk dealer's family were suffering from sore throats. The organism was spread by the raw milk; 284 cases and no deaths were reported. In 1941, there were 37 outbreaks, with 1049 cases and 4 deaths.[11] Again, raw milk and raw-milk products or pasteurized milk improperly handled were responsible for most of the outbreaks. The known responsible diseases, in order of number of cases, were septic sore throat, gastroenteritis, staphylococcal food poisoning, undulant fever, diphtheria and paratyphoid fever.

TABLE 1. *Disease Outbreaks Conveyed through Milk and Milk Products in the United States, 1938–1941.**

DISEASE	NO. OF OUTBREAKS	NO. OF CASES	NO. OF DEATHS
Diphtheria	2	36	3
Dysentery	9	813	0
Food poisoning:			
Staphylococcal	25	868	0
Other	22	522	0
Gastroenteritis	13	1326	0
Paratyphoid fever	2	24	0
Scarlet fever	11	187	1
Septic sore throat	18	2512	12
Typhoid fever	50	478	32
Undulant fever	8	119	0
Unknown	3	38	0
Totals	163	6923	48

*Assembled from data collected by the United States Public Health Service.[8–11]

Dublin, Rogers, Perkins and Graves[12] studied milk-borne outbreaks in upstate New York from 1917 to 1941. During these twenty-four years there were 168 outbreaks, with 9982 cases, of which 6812 were septic sore throat and scarlet fever, 1423 gastroenteritis, 1209 typhoid or paratyphoid fever, 411 bacillary dysentery, 123 diphtheria, and 4 poliomyelitis. Feemster[13] reported on the prevalence of milk-borne disease in Massachusetts from 1933 to 1940; there were 6 outbreaks, with 469 cases. Four outbreaks were of scarlet fever or septic sore throat (412 cases), 2 of undulant fever (12 cases), and 1 of gastroenteritis (35 cases). This is the smallest number of outbreaks recorded for any similar period in Massachusetts. According to Getting,[14] in 1931 65 per cent of the population of Massachusetts lived in communities with regulations requiring the pasteurization or certification of milk. By 1940, 80 per cent of the population were living in these communities, and Feemster[13] states that over 90 per cent of the milk consumed in Massachusetts was pasteurized. Undoubtedly this increase in pasteurization is responsible for the decrease of outbreaks of milk-borne disease.

In 1938 the United States Public Health Service[15] began the collection of data on outbreaks conveyed through foods other than milk and milk products. In that year there were 70 food-borne outbreaks, with 2247 cases and 25 deaths. In one of them, which occurred in Fitchburg, Massachusetts, there were 157 cases of gastroenteritis among 175 persons who ate Greek pastry at a memorial church meeting. In 1939, there were 148 food-borne outbreaks, with 3782 cases and 12 deaths.[16] The foods chiefly responsible, in order of number of outbreaks, were pies and pastries (32 outbreaks), pork and pork products (21), meat and meat products (11), home-canned vegetables, fruits, fish and meat (10), salads (8), sandwiches (7) fowl (7), crab meat (5) and sauces and gravies (5). The commonest diseases as classified by Fuchs[7] were food poisoning (88 outbreaks), gastroenteritis, including diarrhea (88), paratyphoid fever (37), botulism (9), scarlet fever (5) and dysentery (2).

From 1938 to 1941,[15–18] there were 656 food-borne outbreaks, with 17,529 cases and 120 deaths. The commonest diseases in order of numerical importance (Table 2) were food poisoning other than staphylococcal, staphylococcal food poisoning, gastroenteritis, dysentery, typhoid fever, paratyphoid fever and other Salmonella infections, trichinosis, chemical poisoning, botulism, and streptococcal infection.

TABLE 2. *Disease Outbreaks Conveyed through Foods Other than Milk and Milk Products in the United States, 1938–1941.*

DISEASE	NO. OF OUTBREAKS	NO. OF CASES	NO. OF DEATHS
Botulism	25	64	33
Chemical poisoning	7	87	14
Dysentery	16	1038	3
Food poisoning:			
Staphylococcal	208	4865	6
Other	229	6096	7
Gastroenteritis	75	3845	4
Paratyphoid fever (Salmonella infection)	14	475	1
Streptococcal infection	1	40	0
Trichinosis	30	246	14
Typhoid fever	46	578	37
Miscellaneous	5	195	1
Totals	656	17529	120

The responsible vehicles as recorded by Fuchs[15–18] (Table 3), were correlated with the type of disease. Canned foods were the sole vehicle for botulism. Dysentery was most frequently transmitted by an unrecognized food (perhaps owing to a longer incubation period), salads and soups. Chemical poisoning was conveyed by various foods,

including puddings. Food poisoning other than staphylococcal, staphylococcal food poisoning and gastroenteritis were transmitted by nearly all types of foods, the most important for the first two being pastries and ham, and for the third pastries

per cent) of 262 in 1941. Of the 823 outbreaks in the entire period, 80 per cent were transmitted by a food vehicle other than milk and only 20 per cent by milk or a milk product. Moreover, in 1938 and 1939 food-borne outbreaks were more numer-

TABLE 3. *Disease Outbreaks Conveyed through Foods in the United States: Relation between Type of Food and Disease.*

Food	Botulism		Dysentery		Chemical Poisoning		Food Poisoning Other than Staphylococcal		Staphylococcal Poisoning		Gastroenteritis		Paratyphoid Fever		Scarlet Fever		Trichinosis		Typhoid Fever		Other	
	O	C	O	C	O	C	O	C	O	C	O	C	O	C	O	C	O	C	O	C	O	C
Canned food:																						
Commercial	2	5	–	–	–	–	1	3	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Home	23	59	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–
Dressing (stuffing)	–	–	–	–	–	–	3	215	2	128	1	9	1	7	–	–	–	–	–	–	–	–
Fish	–	–	–	–	–	–	14	551	2	20	12	568	1	11	–	–	–	–	4	31	–	–
Gravies, etc.	–	–	–	–	–	–	3	66	4	143	4	75	–	–	–	–	–	–	–	–	–	–
Pork products:																						
Ham	–	–	–	–	–	–	27	226	46	1273	5	192	–	–	1	96	1	7	–	–	2	15
Other	–	–	–	–	–	–	16	389	9	38	3	108	–	–	–	–	27	232	4	61	–	–
Meat, other than pork products	–	–	–	–	–	–	22	603	12	522	13	1367	–	–	–	–	1	5	1	12	–	–
Poultry	–	–	–	–	–	–	30	1270	8	284	5	92	1	21	–	–	–	–	4	77	–	–
Pastries	–	–	–	–	–	–	46	360	75	1365	16	311	–	–	–	–	–	–	1	56	2	49
Puddings, etc.	–	–	–	–	2	73	2	73	1	52	3	33	–	–	–	–	–	–	–	–	–	–
Salads	–	–	3	181	–	–	18	811	9	315	6	121	1	59	–	–	–	–	2	62	3	65
Sandwiches	–	–	–	–	–	–	12	76	10	579	4	116	–	–	–	–	–	–	1	21	–	–
Soup	–	–	1	6	–	–	4	122	1	23	–	–	–	–	–	–	–	–	–	–	–	–
Miscellaneous	–	–	–	–	5	73	11	320	4	177	2	12	–	–	–	–	–	–	14	149	–	–
Unknown	–	–	12	851	1	10	33	690	2	49	12	970	4	305	1	27	–	–	14	100	–	–
Totals	25	64	16	1038	8	156	242	5775	185	4968	86	3574	8	394	2	123	29	244	45	569	7	129

O = number of outbreaks; C = number of cases.

and meats other than ham and pork. Paratyphoid fever was carried chiefly by stuffing, fish, poultry and salads, scarlet fever by ham, trichinosis by pork, and typhoid fever by pork products, fish, poultry and salads. Pastry was responsible for more outbreaks of most diseases than any other known single food; it was usually filled with cream or custard.

The annual increase in the number of recorded outbreaks is due in part to better reporting. As yet, there are several states that are not reporting food-borne disease. Three states reported over half of the outbreaks in 1939. This difference in reporting is not a true indication of the actual prevalence of food-borne disease. The states that reported large numbers of outbreaks are equipped with active epidemiologic units and have made more complete studies.

An analysis of the prevalence of outbreaks for 1938 to 1941, inclusive, reveals that food is a far more prolific source of food-borne disease than is milk alone. Of 112 outbreaks in 1938, 70 (61 per cent) were spread by a food other than milk, and in 1939 the number was 148 (78 per cent) of 189 outbreaks. Food other than milk was the vehicle in 218 (84 per cent) of 260 in 1940, and 223 (85

ous than water-borne, Fuchs[7] recording 91 water-borne outbreaks as compared with 301 where the vehicle was food.

A further example of the prevalence of food-borne disease is furnished by data from Worcester, Massachusetts. During 1941, 2 outbreaks were recorded. In 1942, when the Worcester Department of Public Health became especially interested in food-borne disease, there were 11 outbreaks, with 230 cases. Undoubtedly there were other outbreaks that were not brought to the attention of the department. Assuming that the prevalence of food-borne disease is the same in the remainder of Massachusetts and in the United States as it is in Worcester, there should have been reported a total of 247 outbreaks in Massachusetts and over 7000 in the United States. In short, food-borne disease is more prevalent than is indicated by the reports of health departments.

Food Infection

Amebiasis

Since the Chicago outbreak[19] in 1933, amebic dysentery has ceased to be regarded as a tropical disease. There is no doubt that this disease is more prevalent in the tropics than elsewhere, as

shown by its spread through food handlers among the Americans stationed at Aruba in the Caribbean. Schoenleber[20] found that the stools of over 25 per cent of the men were positive for amebas after several years of residence. An amebic colitis rate of 36.8 per 1000 per annum was reached. Examination of the stools of food handlers revealed that 33 per cent were carriers. Active control of food handlers resulted in a 50 per cent reduction in the incidence of amebiasis after one year, and 92 per cent after three years. The control of food handlers consisted of the examination of the stools of all new employees; those with amebas in their stools were rejected. Stool examinations were reported every month, and if amebas were found, the person was removed from duty, treated with a course of carbarsone or yatren, and returned to duty when three successive stool examinations were negative. Strict hand-washing and fingernail-cleaning routines were instituted. Examination of Italian prisoners[21] from Africa by British Army physicians revealed that asymptomatic carriers of *Shigella dysenteriae* and of *Endamoeba histolytica* cysts were potential sources of epidemics among prisoners. Four carriers who had no symptoms and no recurrence for three to fifteen months were carriers of the former, and two of these were also carriers of the latter.

The Chicago outbreak was the first recognized epidemic of the disease in a civilian population in the United States. At first, the investigators believed that it was due to a high carrier rate among the food handlers of the hotels that were the focus of the epidemic.[22] Of 364 food handlers examined in September, 1933, 11 were carriers and 15 were active cases. In October, the food handlers and other employees were re-examined. There were newly positive stools in 60 of the food handlers and in 100 of 498 other employees. Twenty-three additional carriers were found by culturing the stools. Because the 1400 patients in the two hotels subsequently returned to their homes, cases were scattered among the residents of forty-three states, three Canadian provinces and the Territory of Hawaii. On their return home, forty per cent of the patients were subjected to operation because the illness was mistaken for appendicitis. The mortality rate among all the cases was only 7 per cent. The *Journal of the American Medical Association*[23] published a special review on amebic dysentery as a guide to physicians who were treating patients who had visited the Chicago hotels.

Subsequent investigation[24] revealed that the epidemic was due to infected drinking water. Through faulty plumbing, fresh sewage from another hotel was introduced directly into the drinking water supply of the two hotels. Chlorination was sufficient to kill the pathogenic bacteria, but was not effective against the amebas, which are more resistant to chlorine. In 1935, Hardy and Spector[25] reported another Chicago water-borne outbreak of amebic dysentery among firemen and spectators who drank from a heavily contaminated auxiliary water supply used to extinguish an extensive stockyard fire.

McCoy,[26] of the United States Public Health Service states, "In the World War, bacillary and amebic infections occurred side by side in some military units, and occasionally in the same person." The sources of infection were a human carrier or case, a contaminated water supply, uncooked vegetables from soil fertilized with human excreta, and flies. The same author[27] subsequently wrote that the clinical cases originating in Chicago did not lead to any considerable spread of the disease in communities to which the patients went. He pointed out that control of amebic dysentery by routine examinations of stools of food handlers for the detection of carriers and their subsequent exclusion from food handling did not appear to be practicable on a large scale. Nor is there, he states, any need for the isolation of a case; and, when sanitary disposal of feces is practiced, there is no need for special precautions in the care of stools. The control of amebic dysentery, according to McCoy, depends on the diagnosis and reporting of all cases of dysentery, the recognition of the etiologic agent through adequate laboratory procedures, the education of food handlers in personal hygiene, particularly in washing their hands after visiting the toilet, the examination of stools of food handlers during outbreaks of the disease and the provision of safe water supplies by the elimination of faulty plumbing, cross connections and similar sources of pollution.

Spector[28] reviewed the literature on the results of surveys of infestations with histolytica and described the epidemic prevalence of a small variant of the organism from December, 1933, to summer of 1934 in Chicago. Tonney et al.[29] discuss various laboratory methods used for the diagnosis of amebiasis in the Chicago outbreak and the differential diagnosis of other intestinal protozoa.

Most of the recent surveys indicate that Craig's initial estimate that 5 to 10 per cent of the population are infected with *E. histolytica* is still correct. Tsuchiya and Jean[30] report 2 to 5 per cent of first-year medical and dental students in Missouri as positive. Dienst[31] reports 4 per cent of 245 students at the University of Georgia School of

Medicine as positive, and Faust[32] found 13 cases positive for *E. histolytica* in the post-mortem examination of 202 cases of accidental death in New Orleans.

The treatment of amebic dysentery was discussed at the Cornell University Medical College Conference on Therapy.[33] Two types of medication were said to be effective; the arsenicals, such as carbarsone and hydroxyguinoline (yatren), and alkaloids, such as emetine hydrochloride.

As a consequence of the war, amebic dysentery is likely to awaken new interest in the medical profession. Since it is endemic in temperate zones and is more prevalent, at times even epidemic, in the tropics and subtropics, soldiers may become infected and bring home the disease after demobilization. During World War I, 926 cases of amebic dysentery were reported.[3] Since our troops are more numerous in the present conflict and are in areas where the disease is prevalent, a higher incidence is to be expected.

Trichinosis

Augustine,[34] who studied the problem of trichinosis in New England, points out that until drastic changes are made in the methods of garbage disposal, the only effective means of control must remain with the housekeeper; this means that all pork must be thoroughly cooked before it is eaten. Perkins[35] discusses the prevalence of this disease in New York and points out that the feeding of raw garbage to hogs is the prime factor responsible for the spread of the disease. He cites two recent outbreaks of trichinosis: the first, involving 20 cases among German-Jewish refugees; and the second, 11 cases from food eaten at a church supper.

Wright[36] made a study of the various methods of cooking garbage prior to feeding it to hogs as a means of preventing trichinosis. He discusses the methods used throughout the western and southern portions of the United States, describing the equipment, costs and operation. The costs for fuel and labor varied from 40 cents in the South to $3.50 on the West Coast. This author believes that cooking garbage is a feasible method for the prevention of trichinosis in hogs and thereby in human beings.

An editorial[37] covering progress in the control of trichinosis states that its prevalence in man is proportionate to that in hogs. In Boston, 28 per cent of the adults and 18 per cent of the hogs are infested. In San Francisco, the rates in 1938 were 24 and 15 per cent, respectively, but following the institution of rodent control in the piggeries, the rate of infestation in hogs dropped to 4 per cent in 1941. In the Hawaiian Islands, 14 per cent of wild hogs were trichinous, but when they were kept in concrete or wooden pens and fed cooked garbage, the rate dropped to 0.3 per cent. In Canada, all garbage-feeding hog ranches are required to be licensed and inspected, and all garbage is cooked prior to feeding. Inspection includes rodent control. Swine trichinosis has been reduced to 0.2 per cent.

Witebsky et al.[38] discuss the serodiagnosis of this disease by means of the complement-fixation reaction. The test is sensitive and specific. It can be carried out as a quantitive procedure, and the increase and decrease of the antibody titer can be observed over a period of time.

Outbreaks of trichinosis are usually limited to families, institutions or other units that are fed from the same kitchens. Four cases of trichinosis occurred in March, 1942, in upper New York State.[39] A farmer who purchased a four-month-old pig in the summer of 1941 raised it on cooked food alone. In February, the hog was slaughtered, salted and smoked, and the farmer ate the pork raw in fairly large quantities. On March 3, he became ill and was hospitalized and a diagnosis of trichinosis was made. Three other members of the household became ill with trichinosis, and a dog that had access to the meat developed tenderness of the legs. Two other outbreaks are described by the New York State Department of Health. The first of these involved 7 cases and 2 deaths in a family where a hog fed on kitchen scraps and grain was eaten.[40] Some of the meat was eaten fresh, but more than half of it had been salted and cooked by frying. The second epidemic involved 19 cases and 1 death in four households.[40] The pork responsible for this epidemic was obtained from a garbage-fed pig that had been made into sausages. Several lots of these were given to friends, thereby causing 7 cases among three other households.

Spink and Augustine[41] studied 65 cases in Boston and found that in 46 per cent no relation between the disease and the food eaten was established. They studied the trichinosis infestation of rats; of 193 trapped in large piggeries about twenty miles from Boston, only 2 were infested; of 17 captured in a meat packing house, 5 (30 per cent) were infested; of seventy rats caught in the Boston market or waterfront district, 9 (13 per cent) were trichinous. According to this study, rats do not play an important role in transmitting trichinae to hogs that eat them. Morrison[42] studied trichinosis among patients at the Beth Israel Hospital in Boston. In the seven years preceding 1935, there were 7 cases, in 4 of which the patients admitted having eaten pork. Diagnosis was established by biopsy and by the presence of eosinophilia.

In Maine, Drake et al.[43] studied an outbreak

of 56 cases among 71 persons who ate sausage made by an Italian from the meat of a garbage-fed hog. Ferenbaugh and his associates[44] reported on an outbreak of 64 cases in a CCC camp at Waterbury, Vermont. Roast pork loin, the center of which was undercooked, was traced as the source. The average incubation period was twelve days. Five biopsies revealed 8 to 800 larvae per gram of muscle.

About one third to one half of a company of soldiers developed trichinosis at Camp Edwards in June, 1942, after eating pork products almost daily. The onset was sudden, and the most frequent symptoms were malaise, weakness, dizziness, frontal headaches, aching of muscles of the back and extremities, swelling about the eyes and gastrointestinal disturbances. Marble, Skoog and Bucholz[45] were impressed by the persistent fever, marked prostration, severe headache, muscle pains, swollen eyelids, reddened eyes and tachycardia of the 13 soldiers that were hospitalized. These authors emphasize the fact that inspection of meat does not assure safety from trichinae, and that the only control available to the military personnel is thorough cooking of all pork products.

Typhoid Fever

The typing of *Eberthella typhi* by the method of Craigie and Yen[46–48] has assisted epidemiologists in tracing the source of outbreaks of typhoid fever. Lazarus[49] reports on the typing of 465 cultures from eleven western states. At the present time, unfortunately, this procedure is carried out in only a few laboratories. However, if health officers adopt the typing of strains of *E. typhi*, they can usually ascertain the actual source of epidemics. Eliot and Cameron[50] found the *Vi* agglutination test an aid in epidemiologic investigations of rural typhoid fever.

The isolation of *E. typhi* from stools with different types of mediums was reported by Mayfield and Gober.[51] These workers made 724 isolations, using three mediums as follows: 535 (74 per cent) with lithium chloride Endo agar, 591 (82 per cent) with desoxycholate citrate agar and 601 (83 per cent) with bismuth sulfite agar. They recommend that all three mediums be used routinely, since not all strains were isolated with any one medium.

The New Zealand Department of Health[52] describes a milk-borne outbreak of typhoid fever occurring from February to April, 1942, at Otahuha, near Auckland. There were 26 cases and 4 deaths, and a carrier on a milk farm was responsible. Removal of the carrier as a milk handler and pasteurization of the milk stopped the outbreak. Duff and Hardison[53] describe an unusual meat-borne typhoid-fever outbreak in Cumberland County, Tennessee. Seven cases resulted from souse prepared by a chronic typhoid carrier. *E. typhi* was isolated from the souse three months after its preparation.

Bowman[54] describes 3 outbreaks of typhoid fever from December, 1939, to March, 1940, in Manitoba, Canada, apparently due to cheese made from raw milk and sold without aging. There were 9 cases in the first outbreak, 21 in the second, and 64 in the third. Investigation revealed that two workers at the cheese factory were discharging typhoid bacilli in their stools and that neither had a history of typhoid fever. One of these had apparently been infected by the cheese; his stools became negative in four months. The other was a carrier, and typhoid bacilli were grown from his bile. He had started work in November, 1939, about a month before the outbreaks began. The cheese was allowed to dry for eight days and was sent to the wholesaler on the tenth day. The outbreaks were stopped by removing the carrier as a milk handler and by requiring adequate aging of the cheese. As a result of these outbreaks, Ranta and Dolman[55] studied the survival of typhoid organisms in Cheddar-like cheese. At room temperature, the organisms were detectable for one month; in the refrigerator, survival was prolonged. In one sample, *E. typhi* was recovered seventeen weeks after inoculation. Experiments demonstrated that the typhoid bacillus penetrates cheese by capillary traction.

Bootlegged oysters were responsible for an outbreak of 81 primary and 13 secondary cases of typhoid fever in Louisiana in 1940. Old and Gill[56] traced the outbreak to a chronic carrier who gathered and subsequently infected the oysters, which were sold in the shell and opened by the purchasers.

In New York, a trailer camp was the focus of a typhoid-fever outbreak in 1938.[57] There were 18 cases and 3 deaths. The vehicle was water from a well located in fissured limestone and subject to pollution by nearby cesspools and privies.

Morris et al.[58] report an outbreak of typhoid fever in a group of Negro families in Twiggs County, Georgia, in September, 1940. Two children who died on the twelfth day of "colitis" were the source of the outbreak. The small-colony variety of *E. typhi* was isolated by the authors by employing plating mediums containing utilizable sulfur.

In 1942, Rubenstein[59] reported the occurrence of hospital infections of an intern and nurse from unrecognized cases of typhoid cholecystitis. He advocated the protection of hospital personnel by routine antityphoid inoculation and advised considering all patients with cholecystitis as potential

typhoid carriers. An editorial in the *Journal*[60] estimates that there are 1500 typhoid carriers in Massachusetts, of which only about one tenth are known to the Department of Public Health. It points out that more than 3 per cent of persons who contract typhoid fever continue to harbor the organism, usually in the gall bladder, during the remainder of their lives, and urges physicians to make careful inquiry concerning previous typhoid fever in all cases of cholecystitis. Ames and Robins[61] made a study of the age and sex factors in the development of the carrier state among typhoid-fever patients in New York. Patients over thirty years of age became chronic carriers nine times as frequently as did younger patients. The rate of development of the carrier state at all ages was 2 per cent among males and 4 per cent among females. Sixteen per cent of the female typhoid-fever patients between the ages of forty and forty-nine became chronic carriers. By applying a modified life table based on the experience of New York, these authors estimate the carrier prevalence on January 1, 1940, as 42 carriers per 100,000 population, or 2500 carriers in New York, exclusive of New York City. They estimate that by 1980 the carrier prevalence will decrease to about 200. Four hundred and nineteen (17 per cent) of these estimated carriers were under supervision on January 1, 1940.

There are conflicting reports in the literature concerning the efficacy of sulfaguanidine in the treatment of typhoid carriers. Levi and Willen[62] treated favorably with 0.5 gm. of sulfaguanidine per kilogram of body weight every eight hours for one week a carrier who had had a cholecystectomy prior to the use of the drug. Saphir et al.[63] treated unsatisfactorily 4 carriers with sulfaguanidine over a period of two weeks. Cutting and Robson[64] treated 6 carriers with a variety of drugs and sulfaguanidine, without success. One of these carriers had had a cholecystectomy prior to treatment. Hoagland[65] treated 2 intestinal carriers, one chronic and one convalescent, successfully by large doses of sulfaguanidine. The drug is probably not efficacious in chronic gall-bladder carriers, since such carriers in Massachusetts[66] treated with sulfaguanidine continued to discharge the organism in their stools.

(*To be continued*)

References

1. Whipple, D. V. Ptomaine poisoning doesn't exist. *Hygeia* 19:285, 1941.
2. Dack, G. M. *Food Poisoning*. 138 pp. Chicago: University of Chicago Press, 1943.
3. Dunham, G. C. Military preventative medicine. *Army Medical Bulletin*. No. 23. Third edition. 1198 pp. Carlisle Barracks, Pennsylvania: Medical Field Service School, 1938.
4. Gantenberg, R. Ruhr aus dem Feldzug in Polen, 1939. *Deutsche med. Wchnschr.* 65:1789-1793 and 1820-1825, 1939.
5. Rodenwaldt, E. Die Ruhr in Kriege. *Deutsche med. Wchnschr.* 65:1680-1682, 1939.
6. Wurm, K. Die Ruhr im polnischen Feldzug unter Berücksichtigung. *Med. Klin.* 36:1183 and 1209, 1940.
7. Fuchs, A. W. Disease outbreaks from water, milk and other foods in 1939. *Pub. Health Rep.* 56:2277-2284 and 2468, 1941.
8. *Disease Outbreaks Conveyed through Milk and Milk Products in the United States in 1938*. 7 pp. Washington, D. C.: United States Public Health Service.
9. *Disease Outbreaks Conveyed through Milk and Milk Products in the United States in 1939*. 9 pp. Washington, D. C.: United States Public Health Service.
10. *Disease Outbreaks Conveyed through Milk and Milk Products in the United States in 1940*. 12 pp. Washington, D. C.: United States Public Health Service.
11. *Disease Outbreaks Conveyed through Milk and Milk Products in the United States in 1941*. 11 pp. Washington, D. C.: United States Public Health Service.
12. Dublin, T. D., Rogers, E. F. H., Perkins, J. E., and Graves, F. W. Milk-borne outbreaks due to serologically typed hemolytic streptococci. *Am. J. Pub. Health* 33:157-168, 1943.
13. Feemster, R. F. Milk-borne disease in Massachusetts, 1933-1940. *Am. J. Pub. Health* 31:1169-1173, 1941.
14. Getting, V. A. Progress of milk regulations. *Commonwealth* 25:308-310, 1938.
15. *Disease Outbreaks Conveyed through Foods Other than Milk and Milk Products in the United States in 1938*. 12 pp. Washington, D. C.: United States Public Health Service.
16. *Disease Outbreaks Conveyed through Foods Other than Milk and Milk Products in the United States in 1939*. 26 pp. Washington, D. C.: United States Public Health Service.
17. *Disease Outbreaks Conveyed through Foods Other than Milk and Milk Products in the United States in 1940*. 48 pp. Washington, D. C.: United States Public Health Service.
18. *Disease Outbreaks Conveyed through Foods Other than Milk and Milk Products in the United States in 1941*. 50 pp. Washington, D. C.: United States Public Health Service.
19. Bundeson, H. N., Rawlings, I. D., and Fishbein, W. I. Health hazard of amebic dysentery. *J. A. M. A.* 101:1636-1638, 1933.
20. Schoenleber, A. W. Food handler as transmitter of amebiasis. *J. Trop. Med.* 44:41-43, 1941.
21. Medical Research Council. Dysentery carriers in a prisoners-of-war camp. *Brit. Bull. War Med.* 2:496, 1942.
22. Tonney, F. O., Hoeft, G. L., and Spector, B. K. Threat of amebiasis in food handler. *J. A. M. A.* 101:1638, 1933.
23. Amebic dysentery: a review. *J. A. M. A.* 101:1639-1641, 1933.
24. *Nat. Inst. Health Bull. 166* — Washington, D. C.: Government Printing Office, 1936.
25. Hardy, A. V., and Spector, B. K. Occurrence of infestations with E. histolytica associated with water-borne epidemic diseases. *Pub. Health Rep.* 50:323-334, 1935.
26. McCoy, G. W. Amoebic dysentery: problems presented by outbreak in 1933. *Pub. Health Rep.* 49:141-144, 1934.
27. *Idem*. Control of amoebic dysentery. *Pub. Health Rep.* 4:359, 1934.
28. Spector, B. K. Amebiasis: epidemiology and statistics in Chicago December, 1933, to June, 1936. *Am. J. Pub. Health* 27:694-700, 1937.
29. Tonney, F. O., McIlhenny, M., Hoeft, G. L., and Koonz, C. H. Contributions to laboratory diagnosis of amoebiasis from Chicago outbreak of 1933. *Canad. Pub. Health J.* 26:335-348, 1935.
30. Tsuchiya, H., and Jean, J. T. Incidence of intestinal protozoa among freshmen medical and dental students with especial reference to amebiasis. *Am. J. Trop. Med.* 20:803-808, 1940.
31. Dienst, R. B. Carriers of amebic dysentery among students at University of Georgia School of Medicine. *J. M. A. Georgia* 30:58, 1941.
32. Faust, E. C. Amebiasis in New Orleans population as revealed by autopsy examination of accident cases. *Am. J. Trop. Med.* 21:35-48, 1941.
33. Conferences on Therapy (Cornell University Medical College and New York Hospital). Treatment of some intestinal infestations. *New York State J. Med.* 42:443-451, 1942.
34. Augustine, D. L. Trichinosis and enterobiasis: their importance in New England. *New Eng. J. Med.* 226:488-495, 1942.
35. Perkins, J. E. Epidemiology of food-borne diseases of gastrointestinal tract. *New York State J. Med.* 41:1438-1444, 1941.
36. Wright, W. H. Public health problems concerned in disposal of garbage by feeding it to swine. *Am. J. Pub. Health* 33:208-220, 1943.
37. Editorial. Progress in control of trichinosis. *J. A. M. A.* 116:2855, 1941.
38. Witebsky, E., Wels, P., and Heide, A. Serodiagnosis of trichinosis by means of complement fixation. *New York State J. Med.* 42:431-435, 1942.
39. New York State Department of Health. Trichinosis traced to raw milk. *Health News* 19:106, 1942.
40. New York State Department of Health. Education of public in trichinosis control. *Health News* 19:76, 1942.
41. Spink, W. W., and Augustine, D. L. Trichinosis in Boston. *New Eng. J. Med.* 213:527-531, 1935.
42. Morrison, H. Trichinosis among Jews. *New Eng. J. Med.* 213:531, 1935.
43. Drake, E. H., Hawkes, R. S., and Warren, M. Epidemic of trichinosis in Maine. *J. A. M. A.* 105:1340-1343, 1935.
44. Ferenbaugh, T. L., Segal, L., and Schulze, H. A. Trichinosis epidemic of 64 cases. *J. A. M. A.* 110:1434-1436, 1938.
45. Marble, A. A., Skoog, A. P., and Bucholz, D. J. Trichinosis: report of outbreak at Camp Edwards, Massachusetts. *Mil. Surgeon* 90:636-643, 1942.

46. Craigie, J., and Yen, C. H. Demonstration of types of B. typhosus by means of preparations of type 11 Vi phage: principles and technique. *Canad. Pub. Health J.* 29:448-463, 1938. Demonstration of types of B. typhosus by means of preparations of type 11 Vi phage: stability and epidemiological significance of V form types of B. typhosus. *Ibid.* 29:484-496, 1938.

47. Craigie, J., and Brandon, K. F. Bacteriophage specific for O-resistant V form of *B. typhosus*. *J. Path. & Bact.* 43:233-248, 1936.

48. *Idem*. Identification of V and W forms of *B. typhosus* and occurrence of V form in cases of typhoid fever and in carriers. *J. Path. & Bact.* 43:249-260, 1936.

49. Lazarus, A. S. Typhoid typing in Western states. *Am. J. Pub. Health* 31:60-64, 1941.

50. Eliot, C. P., and Cameron, W. R. Epidemiological investigation of rural typhoid with aid of Vi agglutination test. *Am. J. Pub. Health* 31:599-604, 1941.

51. Mayfield, C. R., and Gober, M. Comparative efficiency of Endo, lithium chloride Endo, desoxycholate-citrate and bismuth sulfite media for isolation of Eberthella typhosa. *Am. J. Pub. Health* 30:69-76, 1940.

52. Watt, M. H. *New Zealand Department of Health Annual Report, Director General of Health, Year 1941-1942*. P. 2.

53. Duff, P. H., and Hardison, A. E. Meat-borne typhoid outbreak in Tennessee. *South M. J.* 35:139-142, 1942.

54. Bowman, M. Epidemic of typhoid fever due to infected cheese, Manitoba. *Canad. Pub. Health J.* 33:541-547, 1942.

55. Ranta, L. E., and Dolman, C. E. Preliminary observations on survival of *S. typhi* in Canadian Cheddar-type cheese. *Canad. Pub. Health J.* 32:73, 1941.

56. Old, H. N., and Gill, S. L. Typhoid fever epidemic caused by carrier "bootlegging" oysters. *Am. J. Pub. Health* 30:633-640, 1940.

57. Lembecke, P. A., and Rafle, P. J. Outbreak of typhoid fever associated with trailer camp. *New York State J. Med.* 40:1371-1374, 1940.

58. Morris, J. F., Barnes, C. G., and Sellers, T. F. Outbreak of typhoid fever due to small colony variety of *Eberthella typhosa*. *Am. J. Pub. Health* 33:246-248, 1943.

59. Rubenstein, A. D. Unrecognized typhoid cholecystitis as source of hospital infections. *New Eng. J. Med.* 226:722-726, 1942.

60. Editorial. Typhoid carriers. *New Eng. J. Med.* 226:740, 1942.

61. Ames, W. R., and Robins, M. Age and sex as factors in development of typhoid carrier state, and method for estimating carrier prevalence. *Am. J. Pub. Health* 33:221-230, 1943.

62. Levi, J. E., and Willen, A. Typhoid carrier state treated with sulfaguanidine. *J. A. M. A.* 116:2258, 1941.

63. Saphir, W., Baer, W. H., and Plotke, F. Typhoid carrier problem: report of study of 110 carriers at Manteno State Hospital, Manteno, Illinois. *J. A. M. A.* 118:964-967, 1942.

64. Cutting, W. C., and Robson, G. B. Alleged efficiency of medicinal treatment of typhoid carriers. *J. A. M. A.* 118:1447-1449, 1942.

65. Hoagland, R. J. Typhoid carrier state treated with sulfaguanidine. *J. A. M. A.* 120:1211, 1942.

66. Feemster, R. F. Unpublished data.

CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

Weekly Clinicopathological Exercises

FOUNDED BY RICHARD C. CABOT

TRACY B. MALLORY, M.D., *Editor**

BENJAMIN CASTLEMAN, M.D., *Acting Editor*

EDITH E. PARRIS, *Assistant Editor*

CASE 29231

PRESENTATION OF CASE

First admission. A fifty-four-year-old painter entered the hospital because of dyspnea, ankle edema and "severe laryngitis."

Each spring of the three years prior to admission the patient had had an attack of "laryngitis." At the same time he also noted slight dyspnea on exertion, slight ankle edema and abdominal swelling. Thirteen months prior to admission his physician treated him with digitalis. One month later he entered another Boston hospital, where he gave a long history of alcoholism, often drinking more than a pint of whisky a day. After an abdominal paracentesis he felt much better. He was found to have a positive blood Hinton test, the spinal fluid had a protein of 37 mg. per 100 cc. and showed a positive Hinton test, a negative Wassermann test and a midzone rise in the gold-sol curve. He was started on antisyphilitic therapy and was told to eliminate alcohol. Accordingly he limited himself to four glasses of beer a day. After discharge he was followed in the syphilis clinic, where he received alternate courses of Mapharsen and bismuth subsalicylate. Spinal fluid obtained six months prior to admission showed a positive Hinton test but a negative gold-sol curve. During the next five months dyspnea on exertion became so severe that the patient was unable to climb two flights of stairs without resting several times. Four weeks prior to admission he developed marked hoarseness and a "cold" characterized by coryza and cough productive of half a cupful of yellowish sputum a day. During the next two weeks he noticed bits of red blood in his sputum. Simultaneously he began to have frequent epistaxes, although his blood pressure was said to have been 150 systolic, 60 diastolic. The cough gradually improved but his voice remained weak and hoarse. Topical sprays did not relieve the difficulty. One week before admission dyspnea became marked, orthopnea developed, and the ankles and lower legs became quite swollen. At no time did he have precordial pain, palpitation, jaundice, hematemesis, or bloody, tarry or acholic stools.

The family history was noncontributory. At the age of twenty he had had typhoid fever, but this cleared without apparent sequelae. He had a questionable penile sore thirty-one years before entry.

Physical examination disclosed an acutely ill, dyspneic man whose face was mottled and purplish red and whose lips and nailbeds were moderately cyanotic. The neck veins were moderately distended. The pupils were equal and small and reacted to light. Small hemorrhages were seen in the inferior portion of the right fundus. The tongue was red, and smooth at the edges. The pharynx was markedly congested. Expansion of the chest was poor on the right. Over the lower two thirds of the right chest posteriorly and in the right axilla there were dullness to flatness on percussion and diminished to absent tactile fremitus and breath sounds. Grocco's triangle could be outlined readily by percussion. Many moist rales were audible at the left base and in the left axilla. The left border of the heart was percussed 3 cm. beyond the left midclavicular line. The sounds were of fair quality. The aortic second sound was greater than the pulmonic. A harsh systolic murmur was audible at the base of the heart, best heard in the pulmonic area. No diastolic murmurs were heard, and there was no widening of the supracardiac dullness. The abdomen was moderately distended. Slight tympany was percussed in the midportion. Slight shifting dullness in the flank and a fluid wave were demonstrated. The liver edge was felt five fingerbreadths below the right costal margin, and the spleen three fingerbreadths below the left costal margin. The prostate was twice normal size, smooth and nontender. There was marked pitting edema of the legs to the knees.

The blood pressure was 210 systolic, 65 diastolic. The temperature was 101.2°F., the pulse 91, and the respirations 30.

Examination of the blood revealed a red-cell count of 5,830,000, with a hemoglobin of 11.5 gm., and a white-cell count of 5750, with 72 per cent neutrophils, 14 per cent lymphocytes, 11 per cent monocytes and 3 per cent eosinophils. The urine was acid and had a specific gravity of 1.008; the sediment contained 30 to 35 white cells per high-power field. A blood Hinton test was positive, and a Wassermann test negative. The stool was watery, brown and guaiac negative. The serum protein was 8.2 gm., the albumin 4.8 gm., the globulin 3.4 gm. and the nonprotein nitrogen 24 mg. per 100 cc. The prothrombin time was 28 seconds (normal, 22 seconds). A bromsulfalein

*On leave of absence.

test showed 30 per cent retention of the dye in the serum. A throat culture was negative for beta-hemolytic streptococci. An electrocardiogram showed normal sinus rhythm, with a rate of 75. The PR interval was 0.14 second; the ST segment was depressed in Leads 1, 2 and 4; the T wave was diphasic in Leads 1 and 2 and low in Leads 3 and 4.

An x-ray film of the chest was unsatisfactory. The cardiac shadow was within the upper limits of normal. The right leaf of the diaphragm and the costophrenic angles were obscured by fluid. The pulmonary and hilar markings were somewhat prominent. The lung fields were clear.

Soon after admission 1150 cc. of yellowish-red cloudy fluid, having a specific gravity of 1.008, was removed from the right pleural space. The vital capacity was 2.4 liters. After digitalization and the use of diuretics the patient seemed markedly improved but his temperature continued to range between 99.4 and 101.2°F. each day, and the hoarseness continued. On the ninth hospital day he coughed up a small amount of blood and his voice was practically gone. Examination of the larynx after topical cocaine anesthesia disclosed that the right vocal cord was nearly immobile and that the left moved poorly. Both were coated with dried mucus. The mucous membrane of the entire oral cavity was markedly congested.

X-ray examination of the pharynx and hypopharynx showed no evidence of disease, but there was some delay in emptying. An x-ray film of the chest nine days after admission showed that the fluid in the right pleural cavity had increased since the initial examination.

Another chest tap on the twelfth hospital day recovered 175 cc. of dark-red fluid, which contained 200,000 red cells per cubic millimeter. No tumor cells were seen.

Films after thoracentesis revealed the presence of encapsulated fluid in the posterolateral portion of the right lower chest. In addition there was a small amount of free fluid. There was evidence of a fracture of at least several weeks' duration, with callus formation, involving the right eighth rib in the anterior axillary line. There was also an additional fracture of the tenth rib in a similar position, with only a small amount of callus formation about the rib.

The patient gradually improved on supportive treatment, although the hoarseness remained unchanged, and was discharged on the twenty-sixth hospital day.

Final admission (two months later). The patient re-entered the hospital because of sudden pain in the right chest.

Since his last admission the hoarseness had become worse, and exertional dyspnea and cough, occasionally productive of small amounts of blood, continued but did not incapacitate the patient. An x-ray film of the chest, in the Out Patient Department, one month prior to admission, showed persistence of considerable encapsulated fluid in the right posterior chest. Callus was seen about both of the rib fractures. During the month prior to admission the patient had a rather marked swelling of both lower legs, and an ulcer developed over the right medial malleolus. Five days prior to admission he experienced a sudden, severe, sharp pain in the right chest. This persisted to the time of admission and was aggravated by deep breathing. The patient felt feverish and had several shaking chills. Breathing became progressively more difficult and wheezing in character. Two days before entry he began to cough up greenish sputum faintly tinged with blood.

Physical examination disclosed a large, well-developed, well-nourished, red-faced, slightly cyanotic man who coughed frequently, bringing up small amounts of rusty sputum. He breathed with a slight expiratory grunt, and his voice was hoarse and weak. By percussion the heart was enlarged to the left anterior axillary line. The scleras were moderately icteric. The right pupil was irregular in shape and reacted poorly to light. There were flatness and diminished to absent breath sounds over the lower right chest posteriorly. The right upper lung field posteriorly and the entire right lung anteriorly were dull to percussion. Breath sounds were reduced and moist rales were audible in this area. The left lung was entirely clear. The abdomen was moderately protuberant, and the nontender edge of the liver was felt three fingerbreadths below the right costal margin. The tip of the spleen was felt below the left costal margin. Shifting dullness was present in the flanks. There was a 3-cm. oval varicose ulcer over the medial malleolus of the right foot. Homans's sign was negative bilaterally.

The blood pressure was 170 systolic, 85 diastolic. The temperature was 103°F., the pulse 110, and the respirations 35.

The red-cell count was 3,380,000, with a hemoglobin of 10.5 gm., and the white-cell count 10,000, with 96 per cent neutrophils. The urine was acid, had a specific gravity of 1.014 and gave a ++ test for albumin; the sediment contained an occasional white cell and red cell per high-power field.

A roentgenogram of the chest showed diffuse hazy density overlying the entire right lower lung field. There was a localized area of increased density at the base of the right upper lung field.

On the second hospital day, coarse and medium moist rales became prominent on both sides of the chest. The white-cell count was 13,000. The patient was given Mercupurin and sulfadiazine without any apparent response. Cyanosis and jaundice deepened, and incontinence of urine developed. He died approximately forty hours after admission.

Differential Diagnosis

Dr. Reed Harwood: Perhaps we should look at the x-ray films first.

Dr. Laurence L. Robbins: These are the films taken at the time of the first admission. They show some hazy density in the right lower lung field, and slight increase in the vascular markings and the hilar shadows. I think this spot film was taken at that time to demonstrate what I take to be an encapsulated area of fluid.

The fractures that were described are seen here. There is relatively little callus formation. From what I can see I think they could be fractures that were probably not due to metastatic lesions, but I cannot be sure because they were not taken with rib technic. This one in the tenth rib appears to be an ordinary traumatic fracture that is healing normally. The margins of the fracture are somewhat irregular.

Dr. Harwood: Could the fracture have any relation to the encapsulated fluid higher up?

Dr. Robbins: Fractures cause hemorrhage in the pleural cavity but I am not at all sure that that is what happened in this instance.

These are the films that were taken, I presume, a month before the second admission, and here again we have evidence of fluid, which is probably free, with a suggestion of an encapsulated area in this region. In the film taken on the second admission, there is a definite area of density in the right upper lobe, with increase in the amount of fluid. I am suspicious of something in the root of the right hilus.

Dr. Harwood: Is there anything suspicious at the hilus in the film taken at the first admission?

Dr. Robbins: No. The only suspicious hilar shadow is seen on the last film taken.

Dr. Harwood: Do you see any evidence of aneurysm or aortitis?

Dr. Robbins: No.

Dr. Harwood: Do you see any evidence of tumor?

Dr. Robbins: There are only two things that suggest tumor: the presence of fluid and the fractures. As I said, I am not at all certain that the fractures represent areas of metastasis, but that is a possibility.

Dr. Benjamin Castleman: I have just noticed a statement in the Out Patient Department record that should have been in the abstract of the history. The record reads, "The patient states that in January [which was one month before the first admission] he fell down an elevator shaft but does not know whether he had hemoptysis."

Dr. Harwood: There was adequate trauma to account for the fractured ribs.

This patient had numerous conditions, some of which are perhaps not related to his final illness. I plan to take them up in the reverse order of their importance.

In the first place he had syphilis. From the spinal-fluid findings I believe he had syphilis of the central nervous system. The x-ray films show no aneurysm or widening of the supracardiac shadow, so that I think one can say he did not have clinical evidence of syphilitic aortitis or aortic regurgitation. Syphilis of the liver is a rare disease, as is syphilis of the lung. The treatment that he received probably prevented the development of active syphilitic lesions that would be responsible for the final illness.

Secondly, the patient had evidence of cirrhosis of the liver. This consisted of an alcoholic history, an enlarged liver, an enlarged spleen, ascites and a reduced liver-function test. The patient may have had cancer of the liver. There is no evidence for it, but I cannot exclude it. I simply mention it as a possibility.

Then he had heart disease, the evidence for which, in my opinion, comprises dyspnea, edema, cyanosis, rales at the bases and perhaps also pleural effusion. He improved on digitalis and diuretics. The interpretation of the electrocardiogram depends on whether or not he had had digitalis at the time it was taken. The tracing is consistent with digitalis effect, and the record states that he was digitalized in the hospital. If the electrocardiogram was taken before digitalization, it might be evidence of an old posterior infarction. I am going to say that the patient had hypertensive heart disease with congestive failure.

Other possibilities come to mind only to be excluded. For instance, I see no evidence for endocarditis or cor pulmonale.

In regard to the rib fracture, one must conclude from the revised record that the patient had had an injury and had fractured two ribs, and it is more than possible that part of the disease in the right lower chest was due to the resulting effusion, although I cannot be certain about that. He had two other reasons for effusion in the right chest—congestive failure and, I believe, cirrhosis of the liver, in which pleural effusion is not at all uncommon.

Then we come to the consideration of the respiratory tract. The onset of the final illness probably dates from the time when he caught cold. He had coryza, cough, hoarseness and fever, and the x-ray films show no evidence of parenchymal involvement of the lungs. In the early part of the illness I should have suspected a pulmonary lesion from the fact that he coughed up such large amounts of sputum and blood, but in the absence of x-ray evidence of parenchymal involvement I think one must say that he had a severe infection of the upper part of the respiratory tract. We know that he had inflammation of the pharynx and larynx, and probably of the trachea and the larger bronchial tubes.

Is there an additional note about the bacteriology?

DR. CASTLEMAN: No. They were apparently interested only in ruling out *Streptococcus haemolyticus*.

DR. HARWOOD: It is important to know what the organism was in this case. The respiratory infection did not follow the course of an ordinary cold. In the course of a few weeks one would expect that he would stop coughing and that his voice would clear, but he continued to run a fever and remained hoarse up to the end. Seven days before death he had a sudden onset of pain in the chest, followed by fever, chills, cyanosis and jaundice, and this train of events suggests the possibility of pulmonary infarction. Another possibility is that he had pneumonia as a complication of the severe upper respiratory infection. If infarction, what was the source of the embolus? Was it from a thrombosis in the leg? He had an ulcer on the malleolus and had had, at least at one time, some swelling in the leg, which may have been evidence of thrombophlebitis. However, Homans's sign was negative and I cannot say surely that that was the source of the embolus. This chronic and severe infection in the upper respiratory tract was enough to cause bleeding from the mucous membrane. The bacteria may well have invaded the wall of the trachea or larynx and produced a septic thrombophlebitis in a vein in that region. An embolus from that source could have produced infarction to the right upper lung. My diagnosis is septic infarction of the right upper lung, hypertensive heart disease and congestive failure, cirrhosis of the liver and syphilis of the central nervous system.

DR. JACOB LERMAN: How does Dr. Harwood explain the paralysis of the cord?

DR. HARWOOD: I believe the paralysis was simply edema and congestion from chronic infection.

DR. CASTLEMAN: Did you have anything else in mind, Dr. Lerman?

DR. LERMAN: I was thinking of a patient that I discussed some time ago who had deficiency disease producing diffuse atrophy in the region of the larynx with paralysis.

DR. CHESTER M. JONES: For the sake of the record it may be wise to amend one of Dr. Harwood's statements. Syphilis of the liver is rare in this hospital, but in many places it is relatively frequent. I am sure that it is not rare.

CLINICAL DIAGNOSES

Latent syphilis.
Lobar pneumonia.
Laryngitis (? syphilitic or tuberculous).
Hydrothorax, right.
Cirrhosis of liver (? alcoholic).
Fracture of ribs.
Thrombophlebitis and varicose ulcer, right leg.

DR. HARWOOD'S DIAGNOSES

Septic infarction of right upper lung.
Hypertensive heart disease.
Congestive failure.
Cirrhosis of liver.
Syphilis of central nervous system.

ANATOMICAL DIAGNOSES

Lobar pneumonia, right lung.
Empyema, encapsulated.
Cardiac hypertrophy.
Cirrhosis of liver, alcoholic.
Splenomegaly.
Syphilis of central nervous system?
Chronic laryngitis.
Traumatic fractures of eighth and tenth ribs.

PATHOLOGICAL DISCUSSION

DR. CASTLEMAN: At autopsy this man had a slightly enlarged heart, weighing 390 gm. There was no disease of the aortic valve, and I am still at a loss to explain the elevated pulse pressure. There was no dilatation of the ascending aorta and no evidence of syphilitic aortitis, grossly or microscopically. The right pleural cavity was almost completely obliterated by numerous old adhesions, and in the posterolateral aspect of the lower portion there was an encapsulated empyema containing about 300 cc. of dirty, greenish-gray, bloody, fibrinopurulent fluid. The wall of the cavity measured 5 to 6 mm. in thickness, which is evidence that the empyema was present at the time of the first admission. The whole right lung was completely consolidated and on section presented the uniform reddish-gray granular appearance characteristic of pneumococcal lobar pneumonia. The left pleural cavity was free from fluid and adhesions. There was no embolism or infarction in the lung.

The liver was moderately enlarged, weighing 200 gm., and had a diffuse, almost regular granularity such as one sees associated with the alcoholic type of cirrhosis. Microscopic examination confirmed this impression, large areas of fatty vacuolization and liver cells with characteristic acidophilic hyaline granular network being seen. The spleen was enlarged, weighing 600 gm. There was a small amount of ascitic fluid.

The vocal cords were edematous and granular, and microscopically there was a marked chronic inflammatory process, with lymphocytic and plasma-cell infiltration. It certainly was not a gumma. The laryngologist might call this a chronic catarrhal laryngitis.

The only evidence of syphilis was a granularity of the ependyma in the floor of the fourth ventricle, a finding usually associated with syphilis of the central nervous system.

Dr. William Beckman: Five days is a short time to develop encapsulated empyema. If we date the terminal episode from the cold, it was five days from the time of the second admission.

Dr. Castleman: I believe that the patient had the empyema at the first admission, because it was seen on the x-ray film then, and also because the cavity wall was so thick at autopsy. The cause of this empyema is still in doubt. It may well have been secondary to the rib fractures or perhaps to pneumonia in the lower lobe, which could not be seen on the film because of the overlying fluid.

Dr. Robbins: There was no free fluid in the right pleural cavity at autopsy?

Dr. Castleman: No.

CASE 29232

Presentation of Case

A sixty-two-year-old woman entered the hospital complaining of abdominal pain.

The patient had been in perfectly good health for about six months preceding the onset of the present illness. Five or six months before entry, during the early hours of the morning, the patient had an attack of lower abdominal cramps. She attributed this to beans eaten the preceding evening. The attack was followed by exhaustion, but there was no change in the bowel habits, and no residual pain or soreness, and she ignored the incident. About four months prior to admission a similar attack occurred, and again three weeks after that. All these attacks were characterized by rumblings and pain of varying degree definitely increasing with each attack. The cramps were always in the lower abdomen, below the umbilicus, with no radiation or steady pain. Occasionally the attacks were associated with the vomiting of "bitter bile," but the vomiting was rarely prolonged. The attacks occurred at night or early morning and always on a Sunday. They were followed by exhaustion for a day or so, and after the first two the patient thought the stools were darker, although they were never frankly black or bloody. After the third attack she called her physician, who gave her some "pills." During the fourth attack he saw her and gave her a hypodermic injection; at that time he advised x-ray study. Films were taken about three weeks before admission and she was then recommended to this hospital. The last attack occurred two weeks prior to admission and was accompanied by even more pain than any of the others. During this period she had lost approximately 6 pounds.

Twenty-eight years prior to admission, following her third pregnancy, the patient stated that she was unwell for four years. During the pregnancy her hands and feet became swollen and she was said to have had kidney trouble. She had had pleurisy following the delivery, and was told that she had anemia and a "spot on her lung." She recovered from this without any particular treatment, and four years later she again became pregnant, without complications. She was well until three years prior to admission, when she felt tired and run down; her doctor told her that she had anemia, not pernicious anemia, and treated her with liver injections for some weeks and then with liver and iron pills. She recovered and stopped taking the pills. One year prior to admission she had a similar episode, again successfully treated with liver and iron. She had taken no liver or iron for eight or nine months prior to entry.

Her family and marital histories were not remarkable.

Physical examination showed a well-developed and well-nourished, pale woman in no apparent distress. The tongue was smooth and pale, with reddened atrophic papillas. The heart was not enlarged. The abdomen was protuberant, soft and tympanitic. No fluid wave or shifting dullness was elicited. There were no masses or tenderness.

The blood pressure was 158 systolic, 90 diastolic. The temperature was 99°F., the pulse 80, and the respirations 18.

Examination of the blood showed a hemoglobin of 13.6 gm. and a white-cell count of 6700 with 71 per cent neutrophils. A blood Hinton test was negative. The urine was normal.

Roentgenograms brought in by the patient showed on both the barium-enema film and the six-hour film of the barium meal an area of narrowing, 3 cm. long, in the ascending colon. The sigmoid showed diverticulosis. A chest roentgenogram was negative.

An operation was performed on the eighth hospital day.

Differential Diagnosis

Dr. Horace K. Sowles: The x-ray films and the story are somewhat contradictory. The recurrent pain and crampy rumbling with nausea are typical of small-bowel, rather than large-bowel obstruction. It is a fairly typical story of small-bowel tumor, but such a tumor is very rare; a gallstone obstructing the terminal ileum is commoner than tumor. According to the x-ray films, the lesion is not in the small bowel but in the cecum or ascending colon. So in that respect I should say the x-ray films and the history are contradictory.

The x-ray report does not indicate whether it was a smooth-walled or ulcerated obstruction. If smooth walled it might be suggestive of lymphoma or sarcoma rather than carcinoma or tuberculosis. The patient had a "spot on her lung" twenty-eight years before entry, which is probably of no great significance, although the lesion could have been tuberculosis of the cecum. The anemia was not very marked; we are not told what the blood smear showed. Certainly if she had a carcinoma of the cecum, it had not been there for three years, since they are rapidly growing tumors. I think we can assume that the anemia three years before entry was not due to the present illness. Mild anemia within the year might have been associated with the present lesion, but I am going on the basis that this was probably carcinoma rather than tuberculosis and therefore that the three-year-old anemia was not due to this particular lesion. Of course the patient may not have had a carcinoma of the ascending colon; perhaps the radiologist can tell us more about the films.

Dr. Laurence R. Robbins: These films were taken in another hospital. I know nothing about the fluoroscopy of the patient, but here is the area of narrowing, which is quite localized, with rather irregular margins. I should say that much of it is ulcerated, but I cannot be positive of that from these films.

Dr. Sowles: Could the area of narrowing be in the terminal ileum?

Dr. Robbins: It is quite difficult to be sure, but I should say that the lesion is close to the ileocecal valve. It could be in the ileum.

Dr. Sowles: Of course that would fit in more with the story of pain and cramps than if it were in the cecum or ascending colon. In that case the x-ray films do corroborate the clinical history of small-bowel obstruction. Tumors of the small bowel, such as carcinomas and lymphosarcomas, are extremely rare. I happened at one time to see four within a period of two years, which was very unusual. I looked them up at that time and found that in a period of twenty-five or thirty years they had had only 25 or 30 cases at the Mayo Clinic.* I think the best bet is malignant tumor, probably carcinoma or lymphosarcoma, of the terminal ileum.

Dr. Richard H. Sweet: My interpretation of the patient's symptoms was that her intermittent attacks were possibly due to intussusception of the obvious growth, and that her pain was probably small bowel in origin because of proximity to the ileocecal valve. I thought it was carcinoma. I operated on her and performed a right colectomy. The growth was in the ascending colon, and I did the radical type of right colectomy that one would do for carcinoma. The lesion looked grossly like a carcinoma.

Clinical Diagnosis

Carcinoma of ascending colon.

Dr. Sowles's Diagnosis

Carcinoma of terminal ileum.

Anatomical Diagnosis

Tuberculosis of ascending colon and regional lymph nodes.

Pathological Discussion

Dr. Benjamin Castleman: When opened, the specimen showed about 5 cm. from the ileocecal valve in the ascending colon, an annular moderately ulcerated lesion measuring 5 by 3 cm. The margins were ragged, and the base was granular. Histologic examination showed tuberculosis. The regional nodes, which were soft but not large, also showed tuberculosis.

Dr. Robbins: In the films it does not look as if the lesion were that far from the ileocecal valve. But one cannot be sure of the exact location.

Dr. Sweet: It looked farther away at operation. It appeared to be in the middle of the ascending colon. The ileum showed thickening and hypertrophy, as if it had been working against an obstruction for a long period.

Dr. Castleman: There was some secondary infection and necrosis along with the tuberculosis. The intestinal lesion had very little caseation, but there was caseation in the regional nodes.

Dr. Sowles: I think the pathological findings explain the small-bowel type of pain. The ileocecal valve was evidently incompetent.

Dr. Castleman: It is quite possible that there had been intussusception, which produced some of the pain, although one usually associates intussusception with tumors, especially those of the polypoid type.

*Rankin, F. W., and Mayo, C., 2d. Carcinoma of small bowel. *Surg., Gynec. & Obst.* 50:939-947. 1930.

The New England
Journal of Medicine
Formerly
The Boston Medical and Surgical Journal
Established in 1828

Owned by the Massachusetts Medical Society and Published under the Jurisdiction of the Committee on Publications

Richard M. Smith, M.D., *Chairman*
James P. O'Hare, M.D. William B. Breed, M.D.
Conrad Wesselhoeft, M.D. Oliver Cope, M.D.

Official Organ of
The Massachusetts Medical Society
and
The New Hampshire Medical Society

Editorial Board

Joseph Garland, M.D. Stephen Rushmore, M.D.
George R. Minot, M.D. Henry R. Viets, M.D.
Frank H. Lahey, M.D. Robert M. Green, M.D.
Shields Warren, M.D. Charles C. Lund, M.D.
George L. Tobey, Jr., M.D. John F. Fulton, M.D.
C. Guy Lane, M.D. A. Warren Stearns, M.D.
William A. Rogers, M.D. Dwight O'Hara, M.D.
Chester S. Keefer, M.D.

Associate Editors

Thomas H. Lanman, M.D. Donald Munro, M.D.
Henry Jackson, Jr., M.D.

Walter P. Bowers, M.D., Editor Emeritus
Robert N. Nye, M.D., Managing Editor
Clara D. Davies, Assistant Editor

Subscription Terms: $6.00 per year in advance, postage paid, for the United States (medical students, $3.50 per year); Canada, $7.04 per year, Boston funds; $8.52 per year for all foreign countries belonging to the Postal Union.

Material for early publication should be received not later than noon on Friday.

The Journal does not hold itself responsible for statements made by any contributor.

Communications should be addressed to the *New England Journal of Medicine*, 8 Fenway, Boston, Massachusetts.

ONE HUNDRED AND SIXTY-SECOND ANNIVERSARY

The one hundred and sixty-second anniversary of the Massachusetts Medical Society, held at the Hotel Statler, Boston, on May 24, 25 and 26, was extremely well attended in spite of transportation difficulties and the absence of approximately a fourth of the active members of the Society, who are serving with the armed forces. The total registration was 1275, as compared with 1556 at the meeting in 1942.

At the annual meeting of the Council, the most significant action taken was the approval of the recommendation of the Executive Committee that will permit the Society to acquire additional office space from the Boston Medical Library, with an appropriate increase in the fee for maintenance. At the request of certain fellows of the Society, the Section of Anesthesiology was created. And, finally, the appointment of two important committees was authorized. One will consider the ways and means of establishing a postwar loan fund for the use of commissioned members of the Society on their return to civilian practice, and the other will assist the OPA in matters pertaining to supplementary food rations demanded by the special diets that are necessary for certain patients. The following officers were elected for the year 1943–1944: president, Roger I. Lee, of Boston; president-elect, Elmer S. Bagnall, of Groveland; vice-president, Daniel B. Reardon, of Quincy; secretary, Michael A. Tighe, of Lowell; treasurer, Eliot Hubbard, Jr., of Cambridge; assistant treasurer, Norman A. Welch, of West Roxbury; and orator, Joseph C. Aub, of Boston.

The routine business at the annual meeting of the Society was perfunctory. The secretary, Michael A. Tighe, reported concerning membership, and the president, George Leonard Schadt, gave the customary annual report on the state of the Society. At the close of the meeting, the orator, Edward P. Bagg, delivered the annual oration, "Small Puddles," which was printed in the May 27 issue of the *Journal*.

Mr. James A. Hamilton, of New Haven, Connecticut, president of the American Hospital Association, was the principal speaker at the annual banquet. He discussed the types of medical and hospital care that will prevail in the future and emphasized the need for physicians and others interested in such problems to support wholeheartedly and enthusiastically all plans that are on a voluntary, rather than a compulsory, basis. The Shattuck Lecture was given by George W. Thorn, Hersey Professor of the Theory and Practice of Physic at the Harvard Medical School, his subject being "Physiologic Considerations in the Treatment of Nephritis." Dr. Thorn stressed the importance of providing a normal food-and-water

intake for patients with nephritis. He pointed out that maintenance of an adequate plasma-protein level by the intravenous administration of a concentrated solution of serum albumin permits direct treatment of intercurrent episodes of azotemia, dehydration or acidosis by parenteral-fluid therapy. He also mentioned the occasional development of an extensive sodium chloride deficit in chronic nephritis and the importance of plasma-chloride measurements as a guide in the treatment of this situation.

The general scientific sessions were well attended. The papers on the first morning covered various aspects of the Cocoanut Grove disaster, and those in the afternoon were chiefly concerned with wartime medicine. The speakers on the second day discussed general topics of interest and value to the practitioner.

The luncheon meetings of the sections, although considerably less well attended than in the past, went off smoothly, and the technical and scientific exhibits were attractively set up in the Ballroom and Foyer, a location that would be difficult to improve. The motion-picture program was so popular that the seating facilities were inadequate, a circumstance that can undoubtedly be corrected in the future.

All who attended the meeting will testify that the Committee on Arrangements deserves the highest commendation for its efforts.

A SIMPLE LABORATORY TEST FOR PRIMARY ATYPICAL PNEUMONIA

Some of the difficulties in determining the etiology in cases of so-called "virus pneumonia," that is, the primary atypical pneumonias that are not caused by known bacterial agents, were reviewed in the *Journal* last year,[1] and recent contributions in this field were commented on in the June 3 issue of the *Journal*. At best the isolation and identification of a nonbacterial respiratory pathogen is still a problem for highly specialized research workers, and even in their hands requires long and carefully controlled procedures. The demonstration of a simple clinical laboratory test that may serve to distinguish some of the cases of virus pneumonia is therefore highly welcome. The finding by workers[2] at the Thorndike Memorial Laboratory, Boston City Hospital, of cold agglutinins in many cases of this disease during the past months may prove to serve as such a test.

The fact that hemolytic anemia develops in certain severe cases of primary atypical pneumonia and the difficulties encountered in typing the blood in such cases, preliminary to transfusion, led to studies that showed that both these hematologic abnormalities are related to the finding of reversible cold hemagglutinins in the serum or plasma of these patients. Further search revealed that most of the patients with the prevalent severe and extensive forms of primary atypical pneumonia exhibited this abnormality to a varying degree, although hemolytic anemia was only rarely encountered. The cold agglutinins appear only after the illness has progressed for a few days, and the titer then increases as the disease advances. The maximum titers are found at, or soon after, the end of the febrile stage of the illness, after which the titer drops.

Cold agglutinins are easily recognized. They may be suspected if there is difficulty in blood grouping or in the finding of compatible blood donors when the serum of the patient is mixed with his own or with any other person's erythrocytes. If citrated or oxalated blood of such a person is placed in a refrigerator, the cells clump and appear to form a clot, but the mass of cells can be broken up readily by shaking and the clumping can be completely abolished by warming the blood to 37° C. The clumping of the cells sometimes occurs at room temperature and is occasionally noted during a red-cell count. The appearance of agglutination in the cold, its persistence in the cold after dilution of the serum or plasma, and its complete abolition by heating to 37°C. serve to distinguish this phenomenon from other forms of hemagglutination. These autohemagglutinins and reversible cold agglutinins are independent of blood

grouping, and persist after the isoagglutinins are removed from the serum by absorption. In actual tests and titrations, serial dilutions of serum or of plasma from oxalated blood are mixed with a 1 per cent saline suspension of washed human Group O (IV) erythrocytes or of the patient's own cells, and the tubes are observed after the mixtures have stood in an ice bath for one hour or in the refrigerator overnight. If positive, the tubes are then placed in an incubator or in a water bath at 37° C. to see if agglutination is completely abolished.

The phenomenon is not specific for atypical pneumonia and indeed does not occur in many of the cases that are usually classed as virus pneumonias. It is apparently a rare property that has been previously described only in a small number of cases, which mostly include cases of blood dyscrasias and liver diseases, and has also been associated with thrombosis and other peripheral vascular phenomena. The only other infectious disease regularly concerned with the presence of cold agglutinins is trypanosomiasis, in which they have been found both in human cases and in experimentally infected animals.

The occurrence of cold agglutinins in atypical pneumonias is independent of chemotherapy with sulfonamide drugs. What relation, if any, they have to the etiology of the disease has not yet been determined. The finding has now been corroborated in many clinics[3] but it has been demonstrated only in a small percentage of cases diagnosed as primary atypical pneumonia, although it has been found quite regularly in the extensive and severe forms. The bacterial pneumonias, and most of the other common infections that have been studied, are not associated with cold agglutinins. This test may therefore serve as a simple means of distinguishing one of the many forms of the virus pneumonias.

References

1. Finland, M., and Dingle, J. H. Virus pneumonias. I. Pneumonias associated with known nonbacterial agents: influenza, psittacosis and Q fever. *New Eng. J. Med.* 227:342-350, 1942. II. Primary atypical pneumonias of unknown etiology. *Ibid.* 227:378-385, 1942.
2. Peterson, O. L., Ham, T. H., and Finland, M. Cold agglutinins (autohemagglutinins) in primary atypical pneumonias. *Science* 97:167, 1943.
3. Horstmann, D. M., and Tatlock, H. Cold agglutinins: diagnostic aid in certain types of primary atypical pneumonia. *J. A. M. A.* 122:369, 1943.

MEDICAL EPONYM

Wassermann Reaction

The first descriptive account of this epoch-making procedure appeared in the *Deutsche medizinische Wochenschrift* (32: 745, 1906) in an article entitled "Eine serodiagnostische Reaktion bei Syphilis [A Serodiagnostic Reaction in Syphilis]" by A. Wassermann (b. 1866), A. Neisser (1855–1916) and C. Bruck (b. 1879). A portion of the translation follows:

> This method consists in the mixture of inactivated serum from monkeys previously inoculated with syphilitic material with organ extracts, serum and so forth of syphilitic persons and the addition of complement (fresh, normal guinea-pig serum), allowing a certain period for the latter to be bound. Thereupon it is determined by means of an inactivated specific hemolytic serum, together with the blood corpuscles, whether the complement that was first added has been completely or partially fixed. If this is the case, the fact is shown by complete or partial failure of the blood cells to disintegrate, that is, by an arrest of hemolysis.
>
> We may, therefore, say that we have succeeded in obtaining a specific serodiagnostic reaction with syphilitic material.

R. W. B.

WAR ACTIVITIES

CIVILIAN DEFENSE

Emergency Distribution of Blood Plasma

A circular (Medical Series No. 27) recently issued by the Office of Civilian Defense in Washington reads as follows:

1. The United States Director of Civilian Defense has made arrangements so that the Civil Air Patrol will fly blood plasma supplies into stricken areas in the event of emergencies.

2. In instances where, owing to bombing, fire, tornado and so forth, it becomes necessary to supplement stocks of blood plasma in stricken areas, and where other methods of transportation are not available or are inadequate, the regional medical officer should get in touch with the appropriate wing commander of Civil Air Patrol and request emergency air transportation for the plasma. Wing commanders have been authorized to accept such requests only from the regional medical officers.

3. In such event, the wing commander should be given complete details as to where the plasma is to be picked up and delivered. Arrangements should then be made by the regional medical officer to deliver the plasma to and from the airports.

4. These arrangements will apply for all states except those located within the jurisdiction of the Western Defense Command. In those states it is understood the Western Defense Command has sufficient

nontactical airplanes available to furnish such transportation.

5. Appropriate instructions, which will ensure the efficient operation of this transportation facility, will be furnished to state and local chiefs of Emergency Medical Services by their regional medical officers.

6. The current list of wing commanders, Civil Air Patrol, is being furnished to the regional medical officers.

MISCELLANY

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS FOR APRIL, 1943

Diseases	April 1943	April 1942	Seven-Year Median
Anterior poliomyelitis	3	1	1
Chicken pox	1135	2053	1286
Diphtheria	8	13	13
Dog bite	963	1073	959
Dysentery, bacillary	3	9	7
German measles	8651	1983	157
Gonorrhea	379	311	368
Measles	7285	5512	4012
Meningitis, meningococcal	140	23	7
Meningitis, other forms	11	8	*
Meningitis, undetermined	2	2	*
Mumps	770	2411	1109
Paratyphoid infections	42	5	5
Pneumonia, lobar	359	338	540
Scarlet fever	2582	1686	1172
Syphilis	301	553	514
Tuberculosis, pulmonary	273	232	268
Tuberculosis, other forms	19	14	30
Typhoid fever	3	1	2
Undulant fever	3	4	3
Whooping cough	604	931	798

*Pfeiffer-bacillus meningitis only other form reportable previous to 1941.

Geographical Distribution of Certain Diseases

Anterior poliomyelitis was reported from: Easthampton, 1; Fitchburg, 1; Malden, 1; total, 3.

Anthrax was reported from: Haverhill, 1; total, 1.

Diphtheria was reported from: Arlington, 1; Boston, 2; Brookline, 1; Merrimac, 2; New Bedford, 1; Salem, 1; total, 8.

Dysentery, bacillary, was reported from: Boston, 1; Fort Devens, 1; Waltham, 1; total, 3.

Encephalitis, infectious, was reported from: Andover, 1; Melrose, 1; Weston, 1; Worcester, 1; total, 4.

Lymphocytic choriomeningitis was reported from: Brockton, 1; total, 1.

Meningitis, meningococcal, was reported from: Amherst, 1; Arlington, 1; Ashland, 1; Boston, 38; Bourne, 1; Braintree, 1; Bridgewater, 1; Brockton, 5; Brookline, 1; Cambridge, 10; Chelsea, 1; Dedham, 3; Everett, 2; Fall River, 4; Falmouth, 1; Fitchburg, 1; Haverhill, 1; Hingham, 1; Holyoke, 2; Hopkinton, 1; Marblehead, 1; Marshfield, 1; Medford, 1; Methuen, 2; New Bedford, 1; Newburyport, 1; Northbridge, 1; Norwood, 1; Quincy, 3; Revere, 1; Rockland, 1; Somerset, 1; Somerville, 4; Southborough, 1; Southbridge, 2; Springfield, 2; Stoughton, 2; Sudbury, 1; Sutton, 1; Taunton, 1; Walpole, 1; Waltham, 7; Watertown, 3; Webster, 1; West Bridgewater, 1; Winchester, 1; Woburn, 2; Worcester, 2; Camp Edwards, 8; Fort Banks, 1; Fort Devens, 3; Westover Field, 1; Chelsea Naval Hospital, 3; total, 140.

Meningitis, other forms, was reported from: Boston, 5; Brockton, 1; Cambridge, 2; Greenfield, 1; Pittsfield, 1; Worcester, 1; total, 11.

Meningitis, undetermined, was reported from: Chelsea, 1; Everett, 1; total, 2.

Paratyphoid infections were reported from: Dalton, 1; Danvers, 34; Peabody, 1; Salem, 3; Sandwich, 1; Worcester, 2; total, 42.

Septic sore throat was reported from: Boston, 9; Cambridge, 2; Foxboro, 2; Haverhill, 2; Merrimac, 1; New Bedford, 1; Springfield, 1; Swampscott, 1; Topsfield, 1; Wakefield, 2; Winchester, 2; total, 24.

Tetanus was reported from: Northbridge, 1; total, 1.

Trachoma was reported from: Boston, 2; total, 2.

Typhoid fever was reported from: Chelsea, 1; Dartmouth, 1; Everett, 1; total, 3.

Undulant fever was reported from: Chelsea, 1; North Brookfield, 1; Southbridge, 1; total, 3.

Weil's disease was reported from: Gloucester, 1; total, 1.

Meningococcal meningitis again reached a new high point, with an increase of 10 cases over last month's record number. There was a marked increase in April in the number of paratyphoid infections; 80 per cent of these, however, occurred in one institution. Scarlet fever continued its upward march, surpassing previous records. Measles and German measles attained high levels, the latter reaching a towering figure fifty-five times the seven-year median.

The following diseases, in addition to those mentioned above, exceeded their seven-year medians: anterior poliomyelitis, dog bite, gonorrhea, pulmonary tuberculosis and typhoid fever.

Below the seven-year median were: chicken pox, diphtheria, bacillary dysentery, mumps, lobar pneumonia, syphilis, tuberculosis (other forms) and whooping cough.

BOOK REVIEW

A Short History of Nautical Medicine. By Captain Louis H. Roddis, Medical Corps, United States Navy. 12°, cloth, 359 pp., with 12 illustrations. New York: Paul B. Hoeber, Incorporated, 1941. $3.00.

Captain Roddis of the United States Navy has long been interested in medical history, and a series of his papers on nautical medicine, which have appeared from time to time in the *Annals of Medical History*, are now brought together in book form. A companion volume, not yet published, will consist of a series of biographies of the surgeons general of the United States Navy.

The present book takes up nautical medicine from the earliest time, giving excellent descriptions of diseases and disasters in old sailing ships, the history of the beginning of the medical department in the Navy and the general rise of naval hygiene, particular attention being paid to the United States Navy. There is also a chapter on nautical medicine in the merchant marine, as well as one on uniforms and insignia. In the last chapter Captain Roddis lists the various topics that might be considered under the term of research in nautical medicine.

This is an interesting and well-written book. The facts are chosen with care, for the author is an expert historian. His style, however, is somewhat heavy for popular consumption. There is plenty of romance associated with nautical medicine, which somehow has evaded the author. As a factual treatise on the subject, brief though it is, this book is highly recommended. There are interesting illustrations, but the volume lacks an index.

NOTICES

SOUTH END MEDICAL CLUB

A regular meeting of the South End Medical Club will be held at the headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston, on Tuesday, June 15, at 12 m. A motion-picture film covering the use of material from the adrenal cortex will be shown. Physicians are cordially invited to attend.

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING THURSDAY, JUNE 17

THURSDAY, JUNE 17

*10:00–11:30 a.m. Medical staff rounds. **Peter Bent Brigham Hospital.**

TUESDAY, JUNE 22

2:00–4:00 p.m. Myasthenia gravis clinic. **Out Patient Department, Massachusetts General Hospital.**

WEDNESDAY, JUNE 23

*12:00 m. Clinicopathological conference. **Children's Hospital.**

*Open to the medical profession.

JUNE 15. South End Medical Club. **Notice elsewhere on this page.**

DISTRICT SOCIETIES

MIDDLESEX NORTH

JULY 28.

WORCESTER NORTH

JULY 28.

BOOKS RECEIVED

The receipt of the following books is acknowledged, and this listing must be regarded as a sufficient return for the courtesy of the sender. Books that appear to be of particular interest will be reviewed as space permits. Additional information in regard to all listed books will be gladly furnished on request.

Stitt's Diagnosis, Prevention and Treatment of Tropical Diseases. By Richard P. Strong, M.D., Sc.D., D.S.M., C.B., professor of tropical medicine, emeritus, Harvard University, consultant in tropical medicine to the Massachusetts General Hospital and the Boston City Hospital, member of the Health Council, Commonwealth of Massachusetts, colonel, M.C., Army of the United States. Consultant to the Secretary of War and director of tropical medicine, Army Medical School, Washington, D. C. Sixth edition. 8°, cloth. Vol. 1, 817 pp., with 197 illustrations; Vol. 2, 876 pp., with 199 illustrations. Philadelphia: The Blakiston Company, 1943. $21.00 for the set.

These two volumes constitute an authoritative reference work in the field of tropical medicine. The first five editions of this book were written by Dr. E. R. Stitt. This new sixth edition has been entirely rewritten by Dr. Strong, with the assistance of the faculty of the Harvard Medical School and other authorities. Recent research has been quoted extensively, and bibliographies have been added to each chapter. The prevention of disease has been stressed, so much so that the title of the book has been changed by adding the word "prevention." The sixth edition was first published in 1942; since the demand soon exhausted the issue, a reprint became necessary in 1943.

(Books Received continued on page xi)

Step Along with
BORN 1820
Still going strong
JOHNNIE WALKER
BLENDED SCOTCH WHISKY
THE FLAVOR of Johnnie Walker is different. One small sip tells you that it is a distinctive whisky . . . Scotch at its smooth, mellow best! Distilled and bottled in Scotland. Enjoyed all over America!
Johnnie Walker Red Label
RED LABEL 8 YEARS OLD
BLACK LABEL 12 YEARS OLD
Both 86.8 proof
Canada Dry Ginger Ale, Inc., New York, N. Y. • Sole Importer

64th Year

RING SANATORIUM AND HOSPITAL

ARLINGTON HEIGHTS, MASS.

For the diagnosis, care and treatment of mental, nervous and chronic medical diseases.

Facilities for all accepted therapeutic measures, including artificial fever and shock therapy.

Outpatient department for referred cases only. Moderate, all-inclusive rates.

VOLTA R. HALL, M.D., MARTHA BRUNNER, M.D.
Resident Physicians

HALLAM T. RING, *Administrator*

163 Hillside Avenue Tel. ARL 0081

Dr. Taylor's Private Hospital

For the Treatment of
ACUTE or CHRONIC
ALCOHOLISM

House well equipped and furnished, skilled attendants, good food and comfortable rooms at moderate rates.

Methods of treatment are those proved best after 30 years' successful experience.

FREDERICK L. TAYLOR, M.D.
45 Centre Street, Boston, Mass.
Near Dudley Street Terminal Roxbury District

WOODSIDE COTTAGES
Framingham, Mass.

A sanitarium specially adapted for nervous and convalescent patients who need rest and upbuilding in normal surroundings.
No committed mental cases.
ARTHUR H. WARD, M.D., *Medical Director*

Glenside

JAMAICA PLAIN, BOSTON, MASS.
A small, attractively located sanitarium for nervous, mild mental or chronic illnesses.
MABEL D. ORDWAY, M.D.
6 Parley Vale Tel. ARN 0044

Wiswall Sanatorium

203 Grove Street
Wellesley, Mass.

A small group of attractive buildings for the care and treatment of nervous and mild mental diseases.

Facilities for training and diversion under the direction of a competent therapist.

E. H. WISWALL, M.D.
Superintendent
HALE POWERS, M.D.
Resident Physician
Tel. WEL 0261

WESTWOOD LODGE
Westwood, Mass.

A modern private sanitarium within fifteen miles of Boston, for the treatment of nervous and mild mental diseases; also for those in need of rest under medical supervision. Separate buildings make possible a satisfactory grouping of patients. The buildings, which include one devoted exclusively to occupational therapy, are surrounded by over one hundred acres comprising woodlands, gardens and pine groves.

Superintendent
WILLIAM J. HAMMOND, M.D.

Resident Physicians
SIDNEY M. BUNKER, M.D.
ELSIE S. NEUSTADT, M.D.

Perkins School

LANCASTER, MASS.

Devoted to the scientific understanding and education of children of retarded development.

Five homelike and attractive buildings surrounded by 85 acres of campus and gardens.

Medical Direction Experienced Staff
FRANKLIN H. PERKINS, M.D.

ADAMS HOUSE
(OF ADAMS NERVINE)
Established 1877

A sanitarium-clinic for the psychoneuroses exclusively

Located in beautiful suburban Boston
JAMES MARTIN WOODALL, M.D.
Medical Director
990 Centre Street
Boston (Jamaica Plain), Mass.

Baldpate Inc.

Georgetown, Mass.

GEO 2131 — Boston Office KEN 8100

For the treatment of psychoneuro personality disorders, psychoses, alcohol and addictions.

Psychotherapy is the basis of treatme other methods such as *shock ther malaria* and *fever box* are used w indicated.

Occupation under a trained thera diversions and outdoor activities.

H. C. SOLOMON, M.D., *Psychiatrist*
G. M. SCHLOMER, M.D., *Medical Dire*
H. JANE MONTZINGO, M.D.
EUGENE L. SWAN, M.D.
Resident Physicians

A Request for Change of Addres

Must reach us at least a week befo the date of issue with which it is take effect. Duplicate copies cann be sent to replace those undeliver through failure to send such advan notice. With your new address sure to send us the old one, enclosin if possible, your address label from recent copy.

NEW ENGLAND JOURNAL OF MEDICINE
8 Fenway, Boston, Mass.

CHARLES B. TOWNS HOSPITAL

Serving the Medical Profession for Over 40 Years

FOR ALCOHOLISM AND DRUG ADDICTION EXCLUSIVELY

Definite Treatment -- Fixed Charges -- Minimum Hospitalization

293 CENTRAL PARK WEST, NEW YORK, N. Y. -- Tel: SChuyler 4-0770

(Hospital Literature)

Essentials of Proctology. By Harry E. Bacon, M.D., professor and head of the Department of Proctology, Temple University Medical School and Hospital, head of the Department of Proctology, St. Mary's Hospital, consulting proctologist, Rush Hospital, National Stomach Hospital. Douglass Hospital, Mercy Hospital and Paul Kimball Hospital (Lakewood, New Jersey). With an introduction by Curtice Rosser, M.D., professor of proctology, Baylor University, Dallas, Texas. 12°, cloth, 345 pp., with 186 illustrations. Philadelphia: J. B. Lippincott Company, 1943. $3.50.

This manual is designed especially for the general student, the general practitioner and the general surgeon. It represents a condensed compilation of essential data on each subject in the field of proctology.

Obstetrical Practice. By Alfred C. Beck, M.D., professor of obstetrics and gynecology, Long Island College of Medicine, and obstetrician and gynecologist-in-chief, Long Island College Hospital, Brooklyn. Third edition. 4°, cloth, 938 pp., with 1064 illustrations. Baltimore: Williams and Wilkins Company, 1942. $7.00.

This third edition of a standard text, first published in 1935, has been revised, with the section on operative obstetrics completely rewritten and illustrated, most of the cuts being in color. Details of administration of local anesthesia are discussed in connection with low cesarean section. The artificial interruption of pregnancy is considered, and the requisites for successful forceps extraction are given in detail. A Portuguese edition was printed in 1940.

Doctor in the Making: The art of being a medical student. By Arthur W. Ham, M.B., associate professor of anatomy, in charge of histology, Faculty of Medicine, University of Toronto; and M. D. Salter, M.A., Ph.D., lecturer and research fellow in the Department of Psychology, Faculty of Medicine, University of Toronto. 12°, cloth, 179 pp. Illustrations by Jean McConnell. Philadelphia: J. B. Lippincott Company, 1943. $2.00.

This book is designed primarily for medical students and for those who contemplate becoming medical students. It deals with the motives and mental equipment that are necessary if one is to be a successful medical student, and also discusses the factors that influence the medical student during his school residence.

Renal Lithiasis. By Charles. C. Higgins, M.D., of the Cleveland Clinic, Cleveland, Ohio. 12°, cloth, 140 pp., with 18 illustrations. Springfield, Illinois: Charles C Thomas, 1943. $3.00.

This work is based on the Beaumont lectures delivered by Dr. Higgins in 1942. It represents ten years' study of the etiology and treatment of renal lithiasis, and is designed for ready reference by the student and the established urologic surgeon. All phases of the subject are covered, including the technic of operative procedures and dietary treatment, with tables of diets. Research on the experimental production of renal calculi carried out at the Cleveland Clinic is discussed.

Contribucion al estudio anatomo clinico de las afecciones del endocardio. By Dr. Manual Perea Muñoz. A doctorial thesis (No. 5636) at Universidad Nacional de Buenos Aires, Facultad de Ciencias Medicas. 4°, paper, 363 pp., with 92 illustrations. Buenos Aires: "Esmeralda," 1942.

This pathological study on endocarditis is based on autopsy material available in the Hospital of Ramos Mejía, Buenos Aires, during the period 1936–1941.

Channing Sanitarium

ESTABLISHED 1879

A pleasant country community of cottages built for the homelike care and treatment of nervous patients. Separate cottage for the care of convalescent nonmental patients. Occupational therapy. Tennis court. Sports building.

JACKSON M. THOMAS, M.D., *Superintendent* CLIFFORD G. ROUNSEFELL, M.D., *Resident Physician*

WELLESLEY, MASSACHUSETTS
Telephone — Wellesley 0464

BREAST MILK

may be obtained at the offices and laboratory of

The

Directory for Mothers' Milk

Incorporated

221 Longwood Avenue, Boston
Telephone BEA 5330

Prices will be adjusted to make the milk available to all who need it

Sent packed in ice to all parts of New England

Bournewood Hospital

300 SOUTH STREET, BROOKLINE, MASS.

Established 1884

For a limited number of cases of mental and nervous diseases

POST OFFICE, CHESTNUT HILL

Telephone PAR 0300

GEORGE H. TORNEY, M.D.

Washingtonian Hospital

41 WALTHAM STREET, BOSTON, MASS.
Founded 1841

A non-profit-making, endowed institution, reorganized, for the

MODERN TREATMENT OF MEN SUFFERING FROM ACUTE OR CHRONIC

ALCOHOLISM

JOSEPH THIMANN, M.D, *Medical Superintendent*
MERRILL MOORE, M.D., *Director of Research*

Visits by Psychiatric and Neurological Staff
Consultants in Medicine, Surgery and the Specialties
Rates Moderate
For information: consult the Medical Superintendent
Telephones: HAN 1750 and 1751

1932 **1942**

1/2 LB. NET (227 GM.)
PABLUM
A thoroughly cooked and dried palatable mixed cereal food, vitamin and mineral enriched.
Pablum consists of wheatmeal (farina), oatmeal, wheat germ, yellow cornmeal, powdered beef bone specially prepared for human use, sodium chloride, powdered alfalfa leaf, powdered yeast and reduced iron. Pablum is thoroughly cooked under pressure and dried, with resultant rupture of the starch granules and some dextrinization. Pablum contains thiamine (vitamin B1) and riboflavin (vitamin G) from natural sources, nutritionally important minerals (iron, copper, calcium and phosphorus), is readily digested, low in crude fiber, palatable, convenient and economical to prepare.
REQUIRES NO COOKING
Add milk or water, hot or cold.
Serve with milk or cream.
MEAD JOHNSON & CO.
EVANSVILLE, IND., U.S.A.
PABLUM IS ALSO SUPPLIED IN 1 LB. 2 OZ. PACKAGES
1/2 LB. NET (227 GM.)
PABENA
OATMEAL enriched with vitamin and mineral supplements, thoroughly cooked and dried.
Pabena consists of oatmeal, malt syrup, powdered whey, powdered beef bone specially prepared for human use, sodium chloride, powdered yeast, and reduced iron. Pabena furnishes vitamin B complex, including thiamine, and nutritionally important minerals (iron, copper, calcium, and phosphorus). As a result of thorough cooking and drying, Pabena is easily digested; palatable; convenient to prepare; and economical to use.
REQUIRES NO COOKING
Add milk or water, hot or cold.
Serve with milk or cream.
MEAD JOHNSON & CO.
EVANSVILLE, IND., U.S.A.
KEEP IN COOL DRY PLACE
ACCEPTED
AMERICAN MEDICAL ASSN.
Council on Foods

8 oz.—1 lb. 2 oz. 8 oz. only

PABLUM, the pioneer precooked fortified infant cereal, now has a companion-product: Pabena is a precooked oatmeal cereal, lending variety to the infant's diet and offering the nutritional and convenient features of Pablum.

BOTH continue to be marketed and advertised only to the medical profession. Samples available on physicians' requests.

MEAD JOHNSON & CO., EVANSVILLE, IND., U.S.A.